

Podrid's Real-World

ECGs

**A Master's Approach
to the Art and Practice
of Clinical ECG Interpretation**

Volume 4 Arrhythmias—Part A: Core Cases

Philip Podrid, MD • Rajeev Malhotra, MD, MS

Rahul Kakkar, MD • Peter A. Noseworthy, MD

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Forewords by: Hein J.J. Wellens, MD • Roman W. DeSanctis, MD

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Podrid's Real-World ECGs: A Master's Approach to the Art and Practice of Clinical ECG Interpretation

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Podrid's Real-World ECGs

A Master's Approach to the Art and Practice of Clinical ECG Interpretation

Volume 4 Arrhythmias—Part A: Core Cases

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy, daughter Sapna, and son Sanjay, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

Peter A. Noseworthy

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*Additional cases for further study are available in a separate digital edition,
Volume 4, Arrhythmias—Part B: Practice Cases.*

Foreword

The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven's landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiodiagnostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today's cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled “Real-World ECGs,” by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching

purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient's clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.

continues

These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important component for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, realworldECGs.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of

work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

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Foreword

The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable “workhorse” in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned

from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician's office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of *Podrid's Real-World ECGs* aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in *Podrid's Real-World ECGs* teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

This volume, the fourth in the series, delves into rhythm analysis, covering sinus, atrial, junctional, and ventricular arrhythmias. The

continues

other volumes focus on the basic approaches to reading any ECG as well as on other disease entities for which the ECG is useful:

- Essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the interpretation of all ECGs (Volume 1)
- Atrial and ventricular hypertrophy, acute myocardial ischemia, acute and chronic myocardial infarction, and pericarditis (Volume 2)
- AV and intraventricular conduction disturbances and enhanced AV conduction (Volume 3)
- Narrow and wide complex tachycardias and forms of aberration (Volume 5)
- Miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions (Volume 6)

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs. Every ECG presents a clinical scenario to further enhance the student's

skills at ECG analysis. Importantly, each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: [realworldECGs.com](http://www.realworldECGs.com)

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers optional access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q&A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

Philip Podrid, MD

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Introduction Arrhythmias

The key to rhythm analysis is a comprehensive and organized approach to the ECG, looking carefully at each lead. This approach includes two primary steps: looking for P waves and examining the QRS complexes.

Approach to Rhythm Analysis

Look for P Waves

The P wave is the key to rhythm analysis. Each lead needs to be examined carefully. P waves may not be immediately apparent, and they may be at the end of a T wave or within a T wave or even an ST segment. Since the upstroke and downstroke of the T wave and the ST segment should be smooth, any bumps, notches, or irregularities in the T wave or ST segment could be superimposed P waves. Importantly, look for P waves after any pause in the rhythm. The following questions need to be addressed when analyzing the P waves:

- Are P waves present or absent?
- Is the P-wave morphology normal (sinus rhythm should have positive P waves in leads I, II, aVF, and V4-V6) or abnormal (inverted or biphasic P wave in leads where it should be positive)?
- What is the P-wave (atrial) rate?
- Are the P waves occurring with regular or irregular PP intervals?

- What is the relationship between the P wave and QRS complex? There should be a P wave before or after each QRS complex, and the PR (or RP) interval should be constant. If it is not constant, is it variable with a pattern (*ie*, Wenckebach) or without a pattern (*ie*, AV dissociation)?

Examine the QRS Complexes

Next consider the QRS complexes, addressing the following questions:

- Are the QRS complexes narrow or wide, normal or abnormal?
- What is the QRS complex (ventricular) rate?
- What is the pattern of the QRS complex intervals (*ie*, RR intervals)? Are they regular, regularly irregular (*ie*, irregular RR intervals with a pattern to the irregularity), or irregularly irregular (*ie*, RR intervals that are random without any pattern)?

Importantly, only three supraventricular rhythms are irregularly irregular: sinus arrhythmia, in which there is one P-wave morphology and PR interval; wandering atrial pacemaker or multifocal atrial rhythm (rate < 100 bpm) or multifocal atrial tachycardia (rate \geq 100 bpm), in which there are three or more different P-wave morphologies and

PR intervals without any one P wave being dominant; and atrial fibrillation, in which there is no organized atrial activity or distinct P wave. Polymorphic ventricular tachycardia also has QRS complexes that are irregularly irregular in interval and very variable in QRS morphology. Polymorphic ventricular tachycardia usually has a very rapid rate. Atrial tachycardia or atrial flutter may have variable RR intervals, but there will be a pattern based on the degree of AV block; hence these rhythms are regularly irregular.

Sinus Rhythm

Since the sinus node is located in the right atrium, the sinus P wave is upright in leads I, II, aVF, and V4-V6 and is inverted in lead aVR. The sinus P wave is usually biphasic in lead V1, reflecting right atrial activation (impulse going toward lead V1) followed by left atrial activation (impulse going away from lead V1). There is one P-wave morphology. Although not necessary to establish a sinus rhythm, there is usually a stable PR interval. There are five types of sinus rhythm:

- *Normal sinus rhythm* is a regular rhythm with a stable PP interval at a rate of 60 to 100 bpm.
- *Sinus bradycardia* is a regular rhythm with a stable PP interval at a rate less than 60 bpm.
- *Sinus tachycardia* is a regular rhythm with a stable PP interval at a rate higher than 100 bpm; if seen, rate increase at onset and rate decrease at offset occur gradually.
- *Sinus node reentry* is a regular rhythm with a stable PP interval, usually at a rate higher than 100 bpm. It resembles sinus tachycardia, but the rate increase at onset and rate decrease at offset are abrupt.

- *Sinus arrhythmia* is an irregularly irregular rhythm. The heart rate (PP interval) is variable due to respiration (*ie*, it is a respirophasic arrhythmia). The sinus rate increases with inspiration and decreases with expiration as a result of changing vagal inputs into the sinus node.

Sinus Node Pause

A sinus node pause is identified by a pause in rhythm (long RR interval) with the absence of a P wave during the pause. There are two etiologies for a sinus node pause:

- *Sinus node exit block*: The sinus node generates an impulse on time, but the impulse does not exit the sinus node region to activate the atrium. The duration of the pause (*ie*, PP interval around the pause) is twice the underlying sinus PP interval.
- *Sinus node arrest*: The sinus node fails to develop an impulse. The duration of the pause (*ie*, PP interval around the pause) is unrelated to the underlying sinus rate. It may be shorter or longer than two sinus PP intervals. A pause that is longer than two sinus intervals may be suggestive of sinus node dysfunction (sick sinus syndrome).

Premature Atrial Complex

Premature atrial complex (PAC) is synonymous with premature atrial beat, atrial premature beat, atrial premature complex, premature atrial extrasystole, and atrial premature extrasystole. It has the following characteristics:

- Early (premature) P wave preceding a premature QRS complex. The P-wave morphology and/or PR interval is different than that of sinus rhythm.
- PACs may be unifocal, in which each premature P wave has the same morphology, or multifocal, in which the premature P waves have two or more different morphologies.
- Following the PAC there is a pause of variable duration that is related to the effect of the PAC on sinus node activity. That is, it may not alter the sinus node, it can reset the sinus node, or it may suppress sinus node activity. Therefore, the PP interval surrounding the PAC can be shorter than, equal to, or longer than two PP intervals (FIGURE 1).

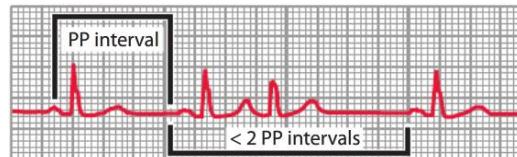
Atrial bigeminy is present when every other QRS complex is a PAC; when every third QRS complex is a PAC, it is termed atrial trigeminy. The presence of bigeminy or trigeminy has no importance and only indicates a repeating pattern. Two sequential PACs is called an atrial couplet; three sequential PACs is known as an atrial triplet or nonsustained atrial rhythm.

Ectopic Atrial Rhythm or Atrial Tachycardia

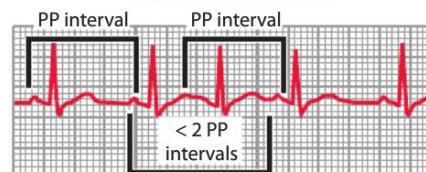
Ectopic Atrial Rhythm

In an ectopic atrial rhythm, the atrial rate is less than 100 bpm. There are distinct P waves of uniform morphology before each QRS complex. The P wave in ectopic atrial rhythm differs from that in sinus rhythm in that it is inverted (negative) or biphasic (negative–positive) in leads where it should be positive. The PR interval is constant and may be the same as or different than that of sinus rhythm. The QRS (RR) intervals are regular.

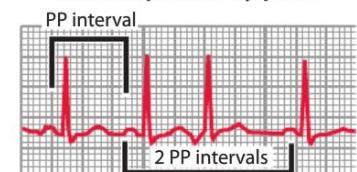
Less than full compensatory pause



Shorter than full compensatory pause (sinus node reset)



Full compensatory pause



Longer than full compensatory pause (possible sinus node dysfunction)

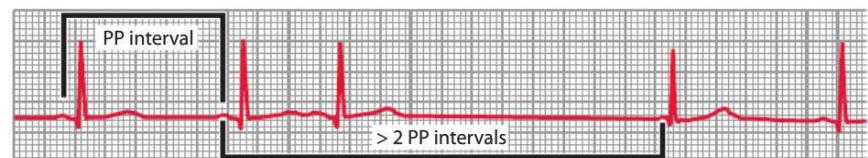


Figure 1. In a premature atrial complex (PAC), the PP interval surrounding the PAC can be shorter than, equal to, or longer than two PP intervals.

Atrial Tachycardia

In atrial tachycardia (ectopic), the atrial rate typically falls between 100 and 220 bpm. The rhythm (atrial rate or PP interval) is generally regular, although it may demonstrate slight irregularity due to changes in automaticity of the ectopic focus. There is a distinct P wave of uniform morphology before each QRS complex. If sequential P waves (without

QRS complexes) are seen (*ie*, when AV block present), then distinct P waves can be seen with an isoelectric baseline between each. The PR interval may be constant or variable if Wenckebach is present. Slightly variable PR intervals may also be seen as a result of antegrade concealed AV nodal conduction, which refers to an atrial impulse that does not completely conduct through the AV node. As conduction velocity through the AV node may variable, some of the atrial impulses conduct entirely through the AV node, some are completely blocked within the AV node, and others conduct partially through the AV node and are extinguished within the node (concealed). Since the AV node is not completely depolarized, it is capable of conducting the next atrial impulse, but at a slower rate. In this situation there is also slight variability of the ventricular rate.

The QRS intervals are regular or may be regularly irregular if variable AV block (*eg*, a variable pattern of 2:1, 3:1, 4:1, 5:1, etc, or even Wenckebach) is present.

Multifocal Atrial Rhythm /Multifocal Atrial Tachycardia

In multifocal atrial tachycardia (atrial rate > 100 bpm) and wandering atrial pacemaker or multifocal atrial rhythm (atrial rate < 100 bpm), there is a distinct P wave before each QRS complex. However, the P-wave morphologies are variable and three or more different P-wave morphologies are present. A dominant P-wave morphology cannot be identified. The PR intervals also vary. The PP and RR intervals are irregularly irregular (*ie*, there is no pattern to the irregularity).

Atrial Flutter

Typical Atrial Flutter

In typical atrial flutter, the atrial rate is usually 260 to 320 bpm and the flutter waves are regular as the mechanism of the arrhythmia is reentry around a fixed circuit, which involves the entire right atrium. The circuit is a result of an area of slow conduction due to fibrosis of the isthmus (*ie*, an anatomic block), which is located between the inferior vena cava and the tricuspid annulus. Hence typical flutter is termed isthmus dependent. The atrial flutter rate may be slower than 260 bpm as a result of anti-arrhythmic drugs or disease of the atrial myocardium; however, the waveforms maintain the typical flutter morphology.

The flutter waves, which are negative/positive in leads II, III, and AVF (due to counterclockwise rotation of the impulse), are uniform in morphology, amplitude, and interval. There is no isoelectric baseline between sequential flutter waves as there is continuous electrical activity. The atrial flutter waves have a continuously undulating (saw-tooth) morphology, reflecting the underlying mechanism of a reentrant circuit resulting in depolarization of the right followed by the left atrium. The QRS complex intervals are regular or regularly irregular if variable AV block (*eg*, a variable pattern of 2:1, 3:1, 4:1, 5:1, etc, or even Wenckebach) is present. In addition, there may be a variable relationship between flutter wave and QRS complex due to antegrade concealed AV nodal conduction (similar to what may be seen with atrial tachycardia). As conduction velocity through the AV node may be variable, some of the atrial impulses conduct entirely through the AV node, some are completely blocked within the AV node, and others conduct partially through the AV node and are extinguished

within the node (concealed). Because the AV node is not completely depolarized, it is capable of conducting the next atrial impulse, but at a slower rate. In this situation there is also slight variability of the ventricular rate.

Atypical Atrial Flutter

In atypical atrial flutter the atrial rate is regular (*ie*, between 320 and 400 bpm). Similar to typical flutter, the mechanism is reentry within the right atrial myocardium. However, there is no anatomic block or area of slow conduction as is seen with typical atrial flutter; hence atypical atrial flutter is not isthmus dependent. In contrast, there are functional changes in membrane refractoriness in a small area of the atrial myocardium that account for the reentrant circuit. Therefore, the circuit is smaller and the velocity of impulse conduction is more rapid as there is only a functional change in refractoriness and not a slowing of conduction as a result of fibrosis. This accounts for the faster atrial rate.

The flutter waves are positive in leads II, III, and aVF (due to clockwise rotation). As with typical atrial flutter, they are uniform in morphology, amplitude, and interval. There is no isoelectric baseline between sequential flutter waves; they are continuously undulating (saw tooth). Similar to typical atrial flutter, the QRS intervals are regular or regularly irregular (if AV block is present). As with typical atrial flutter, AV block may be constant or variable; Wenckebach may also be present. In addition, antegrade concealed conduction may also be present.

Atrial Fibrillation

There is no organized atrial activity or distinct P wave in atrial fibrillation; fibrillatory waves are present. The atrial rate usually ranges from 320 to 450 bpm but can be even more rapid. Fibrillatory waves are usually coarse (> 2 mm) when atrial fibrillation is recent in onset and fine (low-amplitude oscillations) when atrial fibrillation is of longer duration. When coarse, fibrillatory waves may resemble flutter waves (particularly in lead V1); however, fibrillatory waves are irregular in morphology, amplitude, and interval while flutter waves are regular. In addition, QRS complex intervals in atrial fibrillation are irregularly irregular as the conduction to the ventricle is dependent on conduction through the AV node, which will be irregular. The maximum heart rate depends on AV nodal conduction; generally the ventricular rate reaches 170 bpm when the AV node is normal and when no AV nodal blocking agents are being used. Ventricular rates faster than 200 bpm generally reflect an increase in AV nodal conduction velocity, usually a result of increased sympathetic tone or an increase in circulating catecholamines. Ventricular rates less than 100 bpm result from enhanced vagal tone, use of an AV nodal blocking agent (digoxin, β -blocker, or calcium-channel blocker), or intrinsic AV nodal disease.

Atrioventricular Nodal (Junctional) Rhythms

In AV nodal rhythms there is no P wave in front of the QRS complex. An inverted or retrograde P wave (most importantly in lead aVF, which is perpendicular to the atria) may be present following the QRS complex as a result of ventriculoatrial (VA) conduction. The RP interval is

usually stable. However, retrograde or VA Wenckebach may be present, presenting with progressive prolongation of the RP interval and ultimately complete VA block (with the absence of a P wave). The QRS complex intervals are regular, and the QRS complex morphology is similar to that of sinus rhythm, although a rate-related aberration (right bundle branch block, left bundle branch block, or intraventricular conduction delay) may be present.

Premature Junctional Complex

A premature junctional complex (PJC), also termed premature junctional beat, junctional premature complex, or junctional premature beat, is an early QRS complex that resembles the sinus QRS complex but without a preceding P wave. There may be a retrograde P wave that follows the QRS complex (*ie*, negative P wave in at least lead aVF, which is perpendicular to the atria, and possibly in lead II). It is possible that the P wave following the PJC is the on-time sinus P wave. When every other QRS complex is a PJC, it is called junctional bigeminy; junctional trigeminy is present when every third QRS complex is a PJC. The presence of bigeminy or trigeminy has no importance and only indicates a repeating pattern.

Junctional Rhythm and Junctional Tachycardia

Junctional rhythm is a continuous series of junctional complexes at a rate less than 100 bpm; a retrograde P wave may or may not be present. On occasion there may be sinus P waves, which are unrelated to the QRS complexes (*ie*, there are variable PR intervals). This represents AV dissociation, and the atrial rate is slower than the rate of the QRS complexes (which are junctional). This is termed an accelerated junctional rhythm.

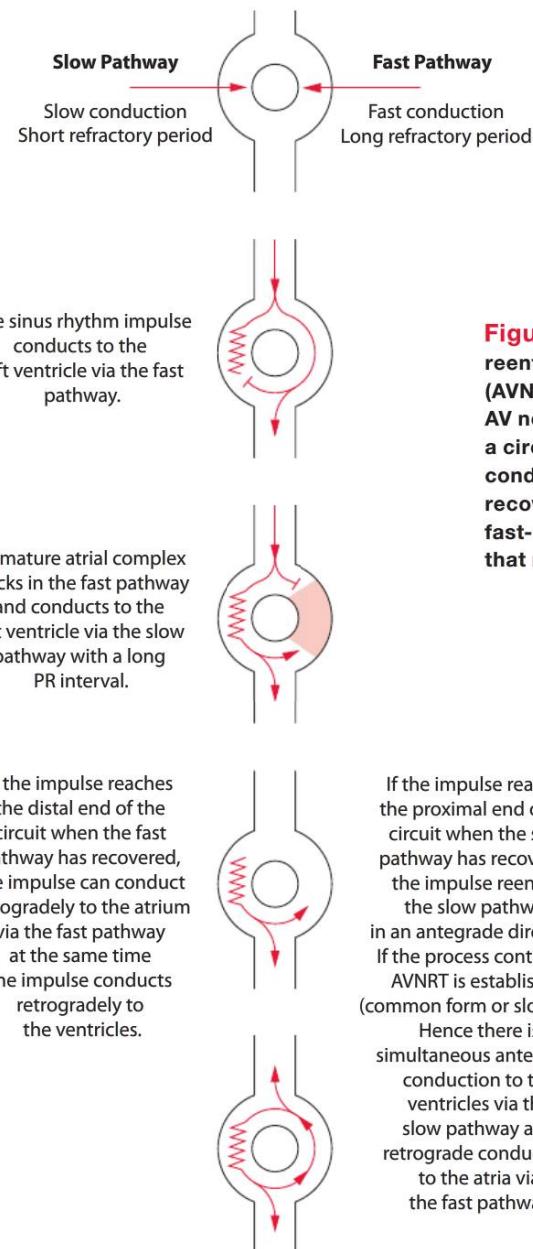


Figure 2. AV nodal reentrant tachycardia (AVNRT) requires dual AV nodal pathways, forming a circuit. There is a slow-conducting pathway that recovers rapidly and a fast-conducting pathway that recovers slowly.

Junctional tachycardia (ectopic) is a continuous series of junctional complexes at a rate exceeding 100 bpm; there is usually a retrograde P wave after each QRS complex and most often there is a short RP interval.

Atrioventricular Nodal Reentrant Tachycardia

Atrioventricular nodal reentrant tachycardia (AVNRT) occurs at a rate of 140 to 220 bpm. AVNRT requires dual AV nodal pathways forming a circuit (via the atrium proximally and the bundle of His distally). There is a slow-conducting pathway that recovers rapidly (short refractory period) and a fast-conducting pathway that recovers slowly (long refractory period) (FIGURE 2).

Typical AVNRT (FIGURES 2 AND 3) is triggered by a PAC occurring when the fast pathway has not recovered and is unable to conduct the impulse antegradely. Therefore, the impulse is conducted antegradely to the ventricles down the slow pathway, which has a short refractory period and recovers quickly. As a result of slow pathway conduction the premature beat has a long PR interval. If the impulse reaches the distal portion of the circuit at a time when the fast pathway has recovered, the impulse can be conducted retrogradely through the fast pathway, activating the atrium retrogradely at the same time that the impulse activates the ventricle antegradely. This is termed slow-fast and in this situation no retrograde P wave is seen (*i.e.*, no RP tachycardia), although in some cases the P wave is superimposed on the end of the QRS complex, appearing to have an R' morphology (in lead V1) or an S wave in the inferior leads (FIGURE 4). Infrequently, typical AVNRT will present with short RP tachycardia (FIGURE 4). This occurs when the fast pathway conducts relatively slowly (as a result of drugs or age-related changes). This is termed slow-slow AVNRT.

Atypical AVNRT (FIGURE 3) occurs when the antegrade conduction to the ventricle occurs via the fast pathway, while the retrograde conduction to the atrium is via the slow pathway. This is termed fast-slow AVNRT and is associated with a retrograde P wave with a long RP interval (long RP tachycardia) (FIGURE 4). It is probable that atypical AVNRT is provoked by a premature ventricular complex (PVC) that arrives at the AV node before the fast pathway recovers and hence is

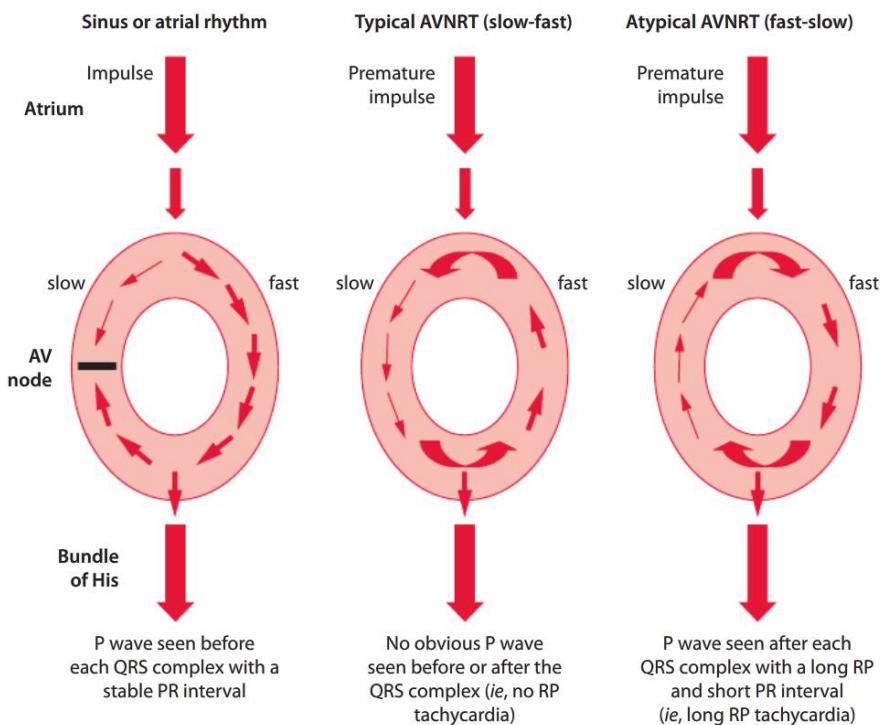


Figure 3. Typical vs atypical atrioventricular nodal reentrant tachycardia (AVNRT).

conducted retrogradely through the slow pathway to activate the atria in a retrograde direction. If the fast pathway has recovered when the impulse reaches the proximal part of the circuit, it will also be conducted antegradely to the ventricles via the fast pathway.

Atrioventricular Reentrant Tachycardia

Atrioventricular reentrant tachycardia (AVRT) occurs in patients with an accessory pathway or a preexcitation syndrome. The rate of AVRT is 140 to 240 bpm. One limb of the circuit is the normal AV node–His-Purkinje system, and the other limb is the accessory pathway. These two pathways are linked proximally via the atrial myocardium and distally via the ventricular myocardium, forming a macro-reentrant circuit. Either limb can conduct antegradely or retrogradely. Hence there is usually a retrograde P wave, generally with a short RP interval (short RP tachycardia) reflecting an increase in the time for conduction through the ventricular myocardium and retrograde conducting pathway (FIGURE 4). Occasionally, a long RP interval may be present (long RP tachycardia).

There are two forms of AVRT: orthodromic and antidromic (FIGURE 5):

- *Orthodromic AVRT* is present when the antegrade conduction to the ventricle is via the normal AV node–His-Purkinje pathway, while retrograde conduction to the atrium is via the accessory pathway. In this situation, AVRT is associated with narrow QRS complexes that have a normal morphology. On occasion, a rate-related aberration may be present, in which case the QRS complexes will have a typical right or left bundle branch block morphology or an intraventricular conduction delay.

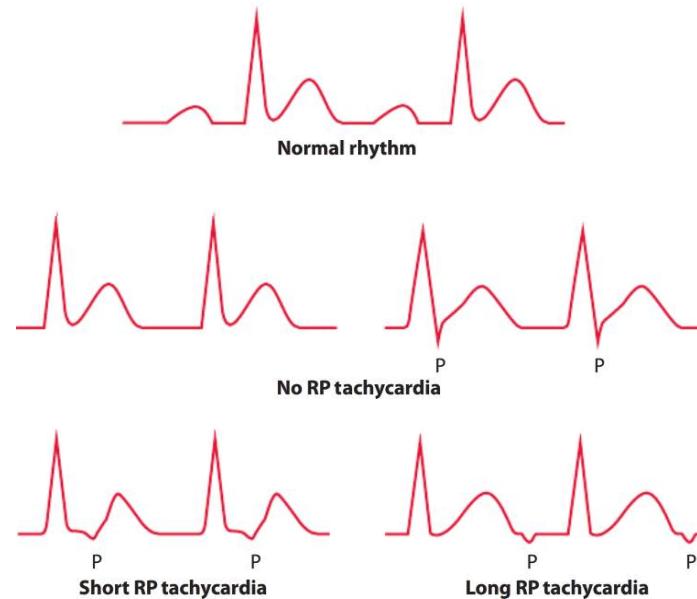


Figure 4. RP tachycardia in atypical atrioventricular nodal reentrant tachycardia.

- *Antidromic AVRT* is present when the antegrade conduction activating the ventricles is via the accessory pathway while retrograde conduction back to the atria is via the normal AV node–His-Purkinje pathway. Since ventricular activation is via the accessory pathway and not the normal His-Purkinje system, there is direct myocardial activation; therefore, antidromic AVRT is associated with wide and abnormal QRS complexes that do not have either a typical right or left bundle branch block morphology. In this situation, the QRS complexes resemble the preexcited complexes during sinus rhythm, although

they may be wider as the QRS complexes are maximally preexcited since ventricular activation is entirely via the accessory pathway rather than representing fusion of conduction via the accessory pathway and the normal AV node–His-Purkinje system (as occurs with the preexcited sinus complex).

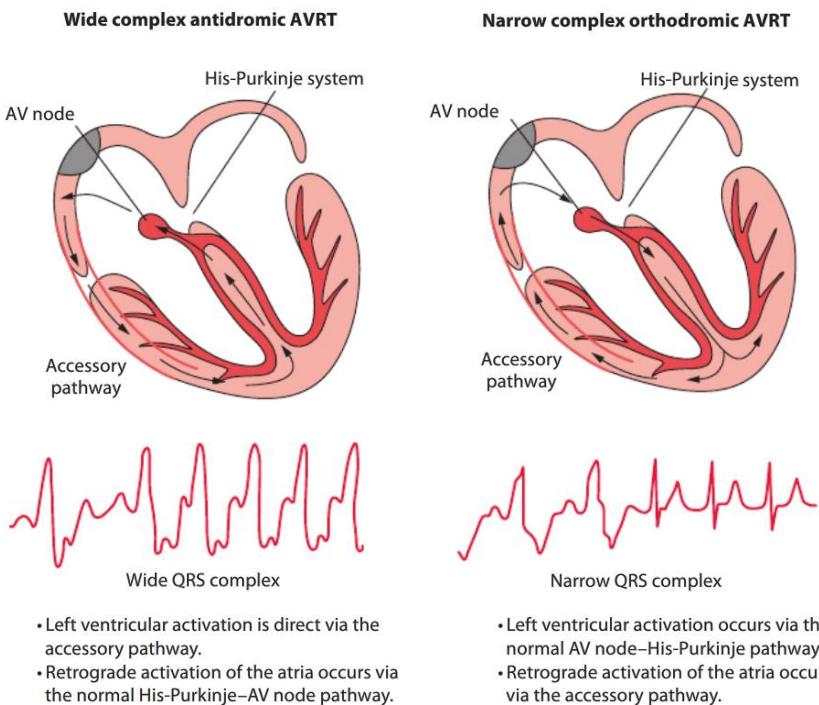


Figure 5. Orthodromic vs antidromic atrioventricular reentrant tachycardia (AVRT).

Ventricular Arrhythmia

Arrhythmia originating from the ventricle is associated with a wide and abnormal QRS complex (≥ 0.12 sec) as ventricular activation is no longer via the normal His-Purkinje system but rather is by direct myocardial stimulation. The QRS complex usually does not have either a typical right or left bundle branch block morphology. P waves may or may not be present. If seen, the P waves may be independent of the QRS complex with variable PR intervals (*ie*, there is AV dissociation). In this situation, the P waves occur at a regular rate that is slower than the ventricular rate and the PR intervals are variable. The presence of AV dissociation during ventricular tachycardia can also be established by the presence of fusion complexes or captured complexes (Dressler complexes). Fusion complexes represent simultaneous ventricular activation via the normal His-Purkinje system from a P wave conducted through the AV node and direct ventricular activation from the ventricular myocardial focus. Hence there is a P wave before the QRS complex with a PR interval that is shorter than the normally conducted sinus complex, and the QRS complex has features of both a sinus complex and a ventricular complex but is different than both. Captured (Dressler) complexes represent ventricular activation due to an atrial impulse (P wave) that is able to penetrate the AV node and completely capture the ventricle during the ventricular arrhythmia, normalizing the QRS complex (*ie*, there is a P wave before the QRS complex and the QRS complex resembles the sinus complex).

Negative P waves may be seen after the QRS complexes if VA (retrograde) conduction is present. QRS complexes and ST-T waves may show

variability in morphology. These changes occur because the ventricular focus generates an impulse that does not activate the ventricles via the normal His-Purkinje system but rather directly through the ventricular myocardium. As a result there may be changes in the ventricular activation sequence and also in ventricular repolarization resulting in variability of the QRS complexes and the ST-T waves. Irregularities of the ST-T waves may also represent superimposed P waves.

Premature Ventricular Complex

PVC, also known as premature ventricular beat, ventricular premature complex, ventricular premature beat, or premature ventricular extrasystole, is a single, early, and wide QRS complex that has an unusual morphology that does not resemble either a right or left bundle branch block. There is no P wave before the QRS complex. A P wave may be seen after the QRS complex; this P wave may be retrograde or it may be an on-time sinus P wave. A full compensatory pause may follow a PVC (*ie*, the PP interval surrounding the PVC is twice the baseline PP interval). This is the result of complete retrograde penetration and total depolarization of the AV node due to the PVC. Hence the AV node is refractory and unable to conduct the next on-time sinus P wave. The subsequent on-time P wave is conducted through the AV node, resulting in a QRS complex (FIGURE 6).

The PVC may be interpolated, in which case it does not alter the underlying sinus rhythm or PP interval; that is, the sinus P wave following the PVC is on time and is conducted through the AV node,

resulting in a native and normal QRS complex that resembles the sinus QRS complex. Hence the PP interval surrounding the PVC is the same as the baseline PP interval (FIGURE 6). However, the PR interval after the PVC may be longer than the baseline PR interval as a result of retrograde concealed VA conduction. In this situation the PVC only partially penetrates the AV node in a retrograde direction and does

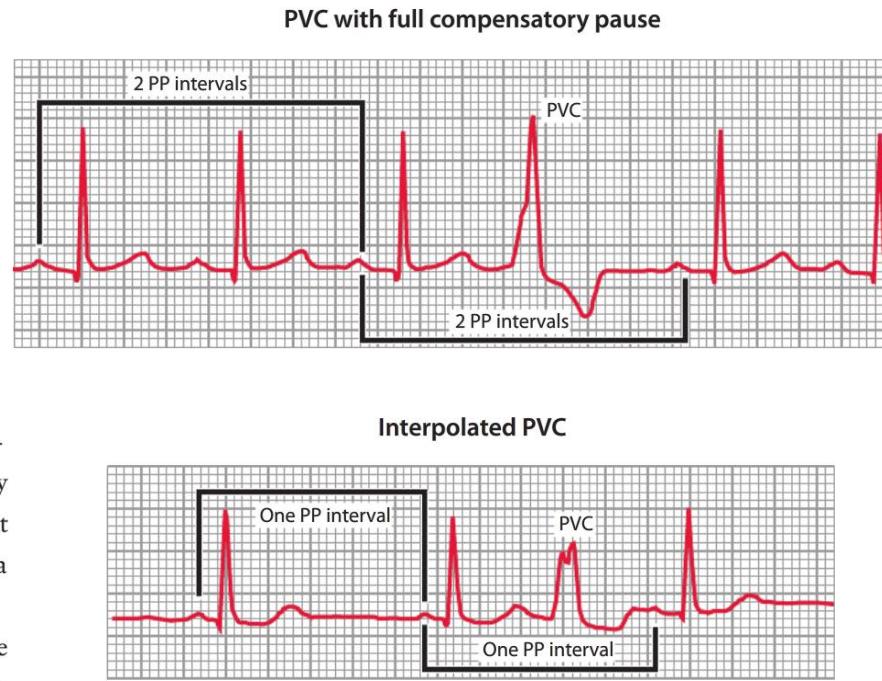


Figure 6. Premature ventricular complexes (PVCs) may be followed by a full compensatory pause or may be interpolated.

not completely depolarize the node (*ie*, the impulse is concealed). Since the AV node is not completely depolarized and not totally refractory, it is able to conduct the next P wave but the rate of conduction through the AV node is slower than normal as a result of partial depolarization and some prolongation of refractoriness, accounting for the longer PR interval after the PVC.

If all the PVCs have the same morphology, they are termed unifocal. If there are different QRS morphologies the PVCs are termed multifocal. Two sequential PVCs is called a ventricular couplet; three in a row is termed a ventricular triplet or may be called nonsustained ventricular tachycardia (NSVT).

When every other QRS complex is a PVC, it is termed ventricular bigeminy; when every third QRS complex is a PVC it is called ventricular trigeminy. The presence of bigeminy or trigeminy has no importance and only indicates a repeating pattern.

Ventricular Rhythm

Ventricular rhythm is the presence of sequential ventricular complexes at a rate of 60 bpm or less. When the rate is 60 to 100 bpm it is termed an accelerated idioventricular rhythm, or it may also be called slow ventricular tachycardia. P waves may or may not be present. If present, the P waves (when seen) will be regular (stable PP interval) but dissociated from the QRS complexes; that is, the PR intervals are variable without any pattern and the atrial rate is slower than the ventricular rate. The P wave may also be retrograde due to VA conduction. In this case the P wave is negative in at least lead aVF (which is perpendicular to the atria) as well as in other leads. Additionally, it will have a fixed

RP interval or coupling interval between the P wave and the preceding QRS complex.

Nonsustained Ventricular Tachycardia (monomorphic or polymorphic)

NSVT is defined as tachycardia (rate > 100 bpm) consisting of three or more sequential ventricular QRS complexes lasting for up to 30 seconds. However, tachycardia may be considered NSVT if it self-terminates. If all the QRS complexes are similar, the NSVT is termed monomorphic. If the QRS complexes have a variable morphology and axis, the NSVT is termed polymorphic. If the QT interval of the sinus QRS complex is normal, the polymorphic NSVT is simply called polymorphic NSVT, which is usually due to ischemia. If the QT interval of the sinus QRS complex is prolonged (*ie*, long QT syndrome), the polymorphic NSVT is called torsade de pointes. This may be due to either a drug that prolongs the QT interval (acquired) or a congenital (genetic) abnormality that results in a channelopathy.

Sustained Ventricular Tachycardia (monomorphic or polymorphic)

Sustained ventricular tachycardia is defined as a series of regular ventricular QRS complexes at a rate 100 bpm or faster that lasts longer than 30 seconds or is terminated in less than 30 seconds (often due to hemodynamic compromise). If all of the QRS complexes have a similar morphology the ventricular tachycardia is termed monomorphic. If the QRS complexes vary in morphology and axis the ventricular tachycardia is termed polymorphic. If the QT interval of the sinus QRS

complex is normal, the polymorphic ventricular tachycardia is simply called polymorphic ventricular tachycardia, which is usually due to ischemia. If the QT interval of the sinus QRS complex is prolonged, the polymorphic ventricular tachycardia is called torsade de pointes.

Ventricular tachycardia that occurs at a rate exceeding 260 bpm is often called ventricular flutter. This is meant only to indicate that the tachycardia is at a very fast rate.

Ventricular Fibrillation

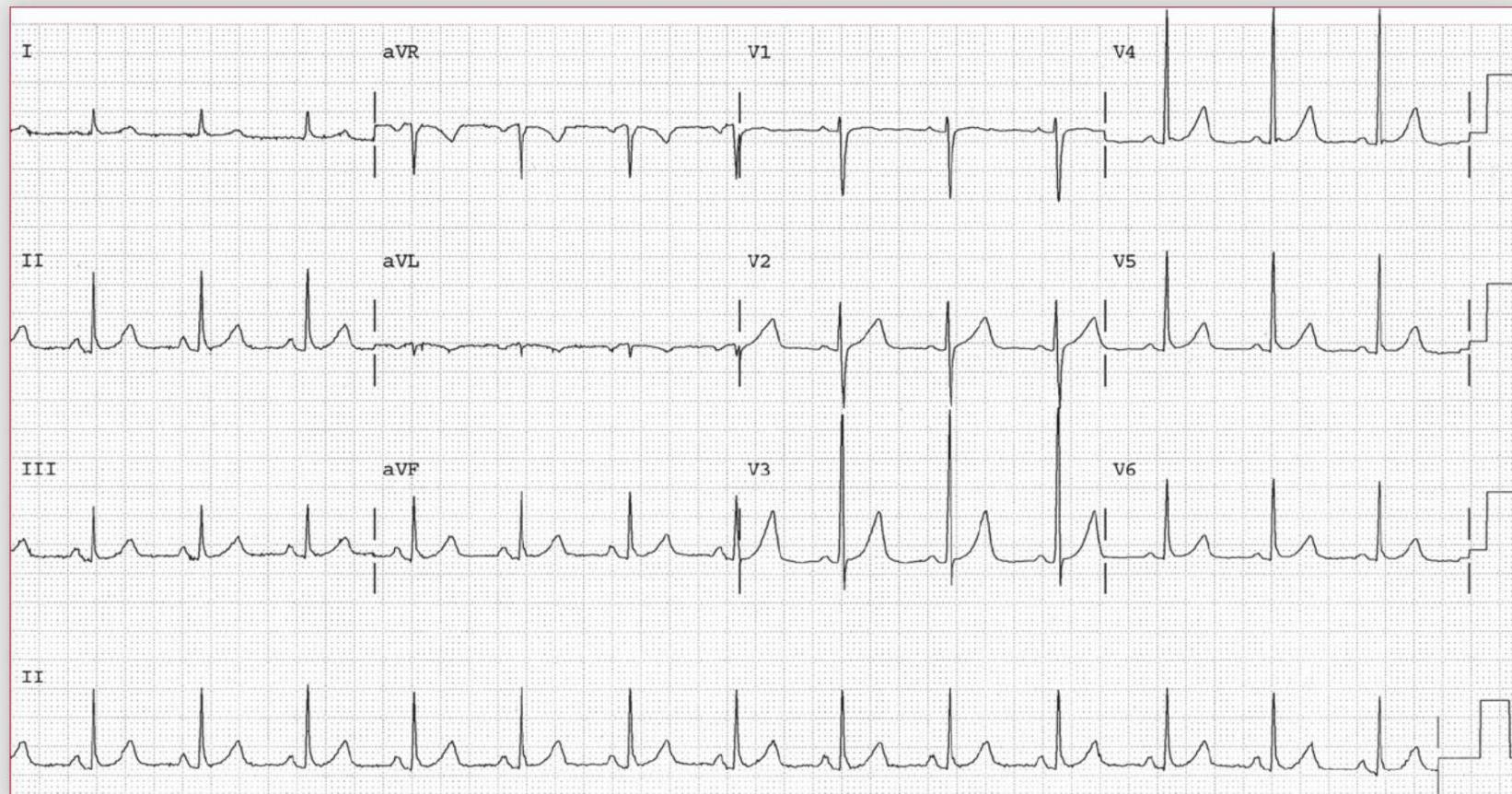
Ventricular fibrillation is identified by the absence of any organized QRS complexes. There are fibrillatory waves that are irregular in morphology, interval, and amplitude. This arrhythmia is most commonly the result of ischemia and can only be terminated with the unsynchronized delivery of a high-energy electrical impulse to the heart, termed defibrillation. ■

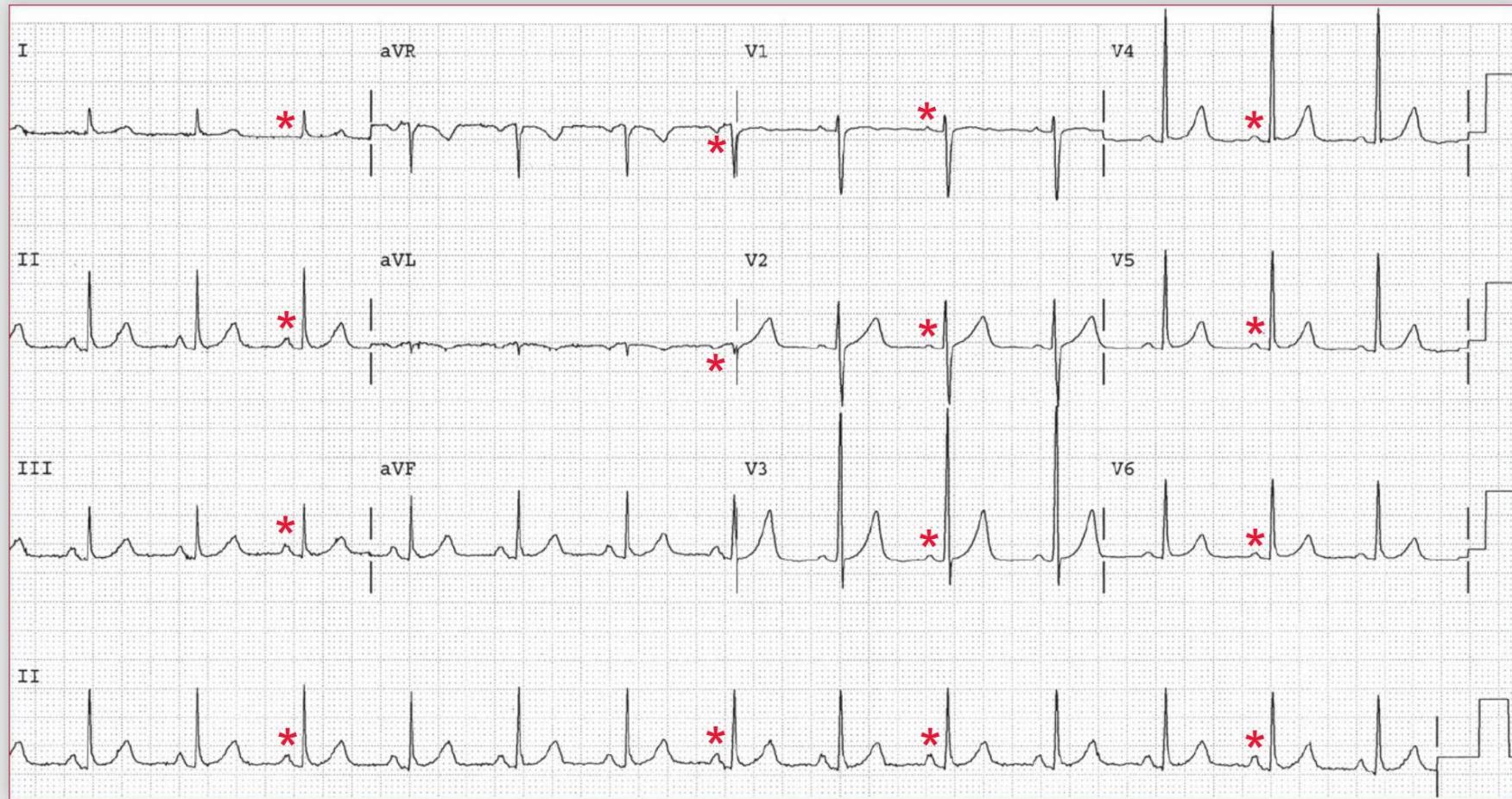
Core ECGs

A 45-year-old man presents to his primary care physician with the complaint of intermittent but frequent palpitations over the past week. He has no significant medical history and is not taking any medications or over-the-counter supplements. He denies any other symptoms associated with the palpitations. Physical examination is completely normal. You obtain the following ECG while the patient is asymptomatic.

What is the diagnosis?

What is the next step in management?





ECG 1 Analysis: Normal sinus rhythm, normal ECG

The rhythm is regular at a rate of 80 bpm. A heart rate between 60 and 100 bpm is normal; rates less than 60 bpm are called bradycardia, and rates over 100 bpm are called tachycardia. There is a P wave (*) before each QRS complex. The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This establishes the rhythm as originating in the sinus node, which is located in the proximal portion of the right atrium. Activation occurring from this structure generates an impulse that is directed from right to left and from up to down. Hence sinus rhythm is associated with a P wave that is upright in leads I, II, aVF, and V4-V6. The P wave of sinus rhythm is inverted in lead aVR (which is the mirror image of the other limb leads). There is only one P-wave morphology. Hence this is a normal sinus rhythm.

The PR interval is 0.16 second, the QRS complex duration is 0.08 second, and the QT / QTc intervals are 380/440 msec. All these intervals are normal. The electrical axis in the frontal plane is normal, between

0° and +90° (positive QRS complex in leads I and aVF). There is normal R-wave progression across the precordium, with transition (R/S > 1) occurring in lead V3. The T waves have a normal morphology (asymmetric with a slower upstroke and more rapid downstroke) and normal axis. Therefore, this is a normal ECG.

Given that the patient's symptoms are intermittent and that he is asymptomatic during the acquisition of this office ECG, the next step in management is to obtain information about the patient's rhythm during a symptomatic episode. A Holter monitor (continuous monitoring for 24 to 48 hours) can be used for frequent episodes (*ie*, more than one in 24 hours), while an event or loop recorder (transtelephonic monitor) is used for infrequent episodes. ■

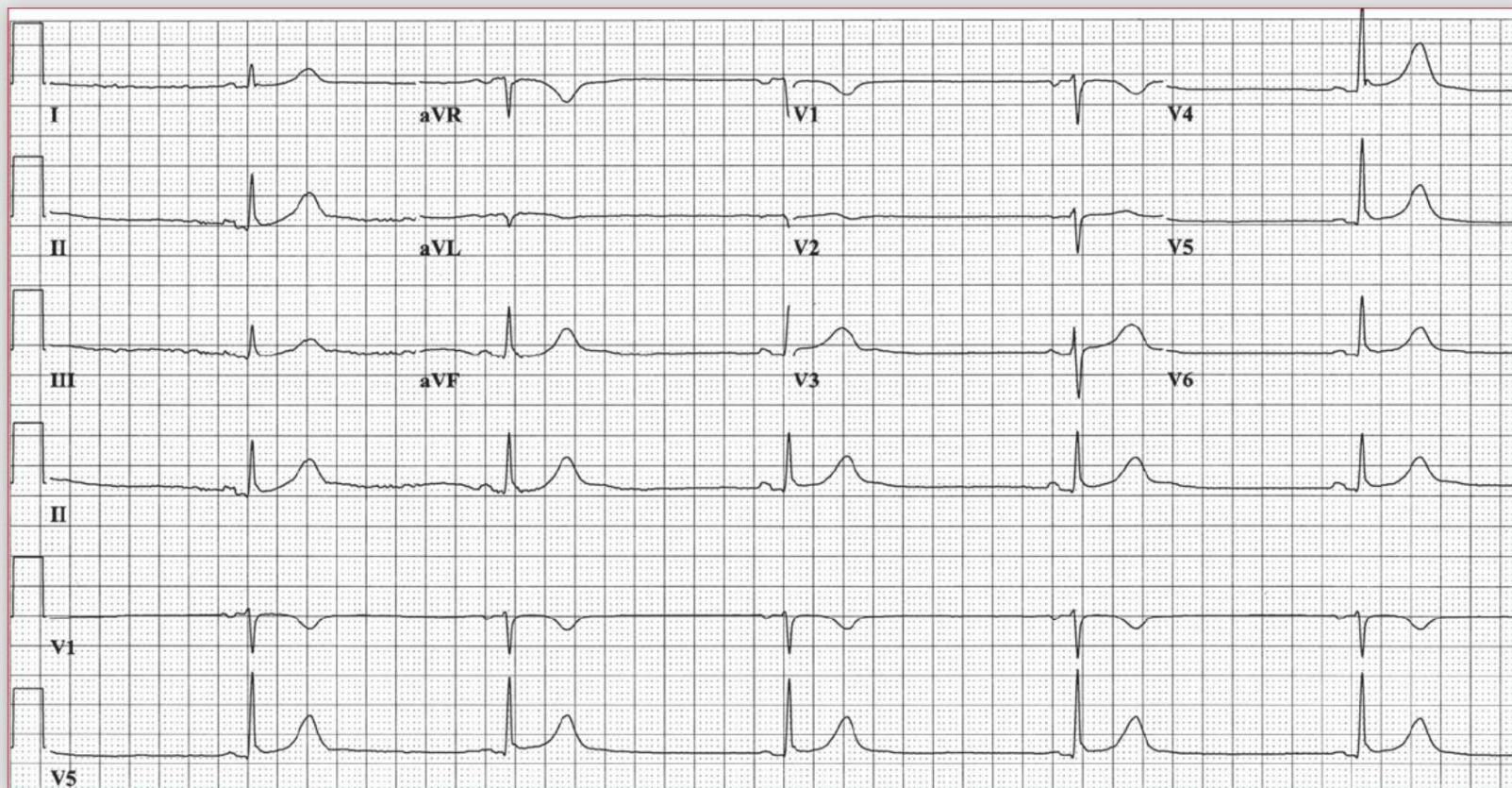
Notes

A 44-year-old woman presents to your office with complaints of dizziness and lightheadedness with exercise for the past 2 years. These symptoms prevent her from leading an active lifestyle. She never experiences these symptoms at rest, and she is not taking any medications. Her physical examination is

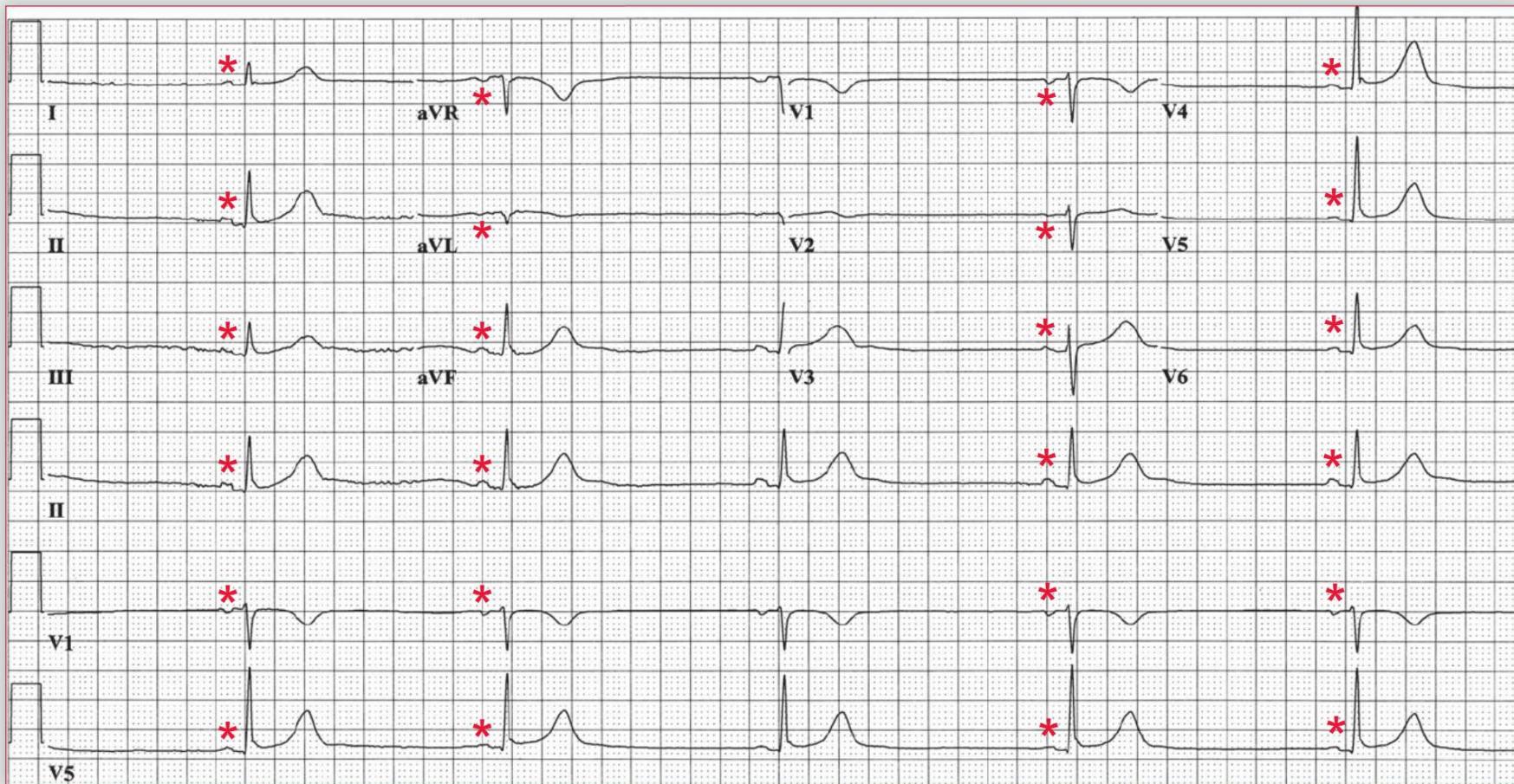
completely normal. You obtain the following ECG in your office.

You then exercise the patient carefully on a treadmill. After 3 minutes of fast-paced walking, she becomes symptomatic. Her blood pressure drops to 84/51 mm Hg, and her heart rate is 56 bpm and regular. Her oxygen saturation remains at a normal level.

What is the clinical diagnosis?
What is the next step in management?



Podrid's Real-World ECGs



ECG 2 Analysis: Sinus bradycardia

There is a regular rhythm at a rate of 32 bpm. There is a P wave (*) before each QRS complex, and it is upright in leads I, II, aVF, and V4-V6. There is one P-wave morphology and a stable PR interval (0.16 sec). This is sinus bradycardia. The QRS complex duration and morphology are normal. The QRS axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (540/390 msec). There is normal R-wave progression across the precordium, and the T waves are normal (asymmetric with a slower upstroke and more rapid downstroke).

The slow heart rate observed in this resting, awake ECG is not sufficient to warrant the placement of a pacemaker in an asymptomatic individual. Many individuals, including well-trained athletes, exhibit bradycardia due to a high degree of vagal tone. Sinus bradycardia is

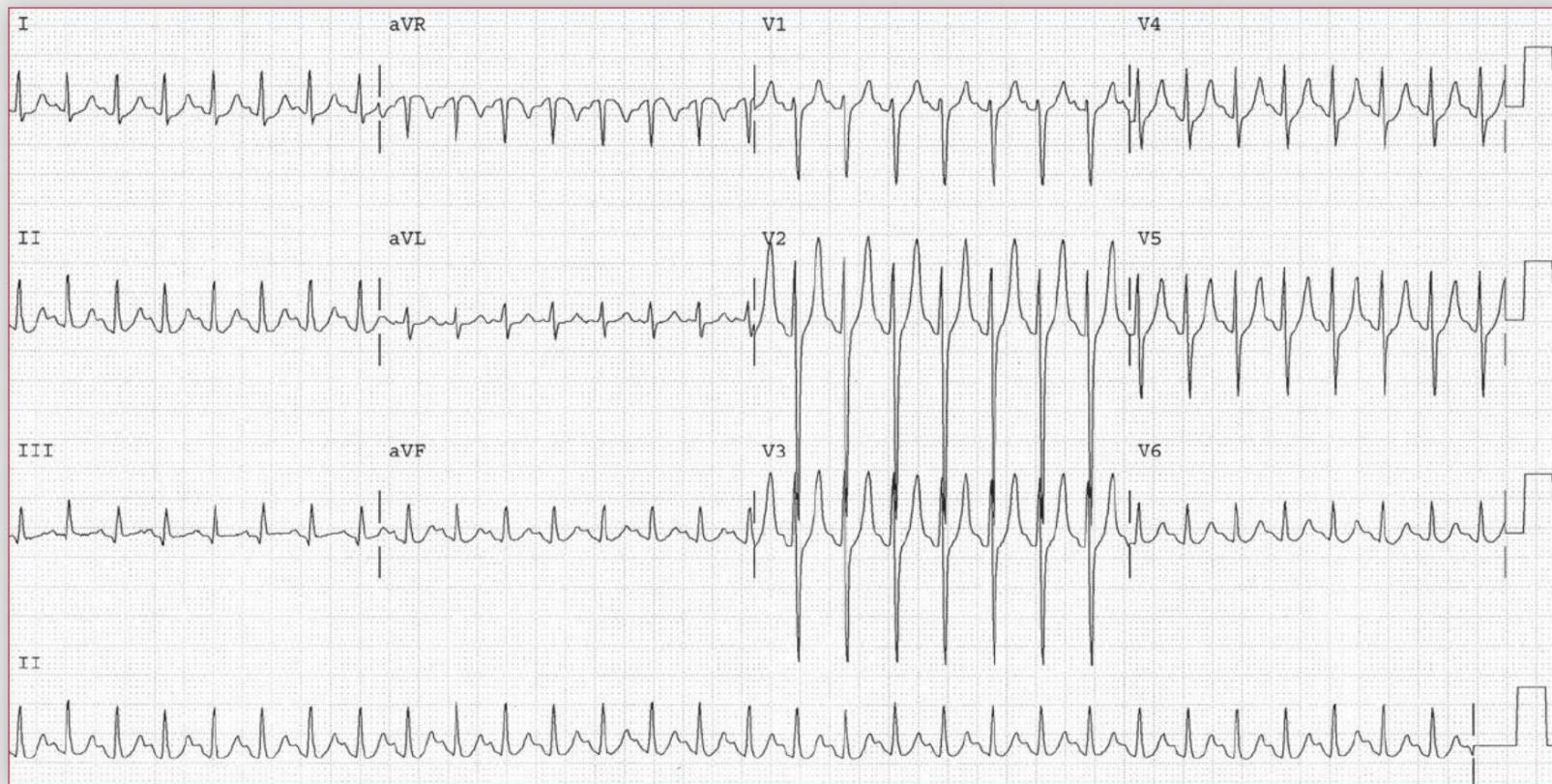
commonly observed at night while patients are sleeping as a result of the increased vagal tone that occurs at this time. In this case, the patient's symptoms occur only with exercise. Noted is the fact that her heart rate only reaches 56 bpm at peak exercise. The inability of the heart rate to increase during exercise in proportion to metabolic demand is termed chronotropic incompetence. The maximum predicted heart rate (MPHR) for any individual is defined by the following equation: $MPHR = (220 - \text{age}) \text{ bpm}$. Various criteria for defining chronotropic incompetence have been used, including less than 85% of MPHR at peak exercise or an absolute cut-off of less than 100 bpm at peak exercise. Symptomatic chronotropic incompetence, as seen with this patient, is a class I indication for permanent pacing. In general, a rate-responsive pacemaker is used. ■

Notes

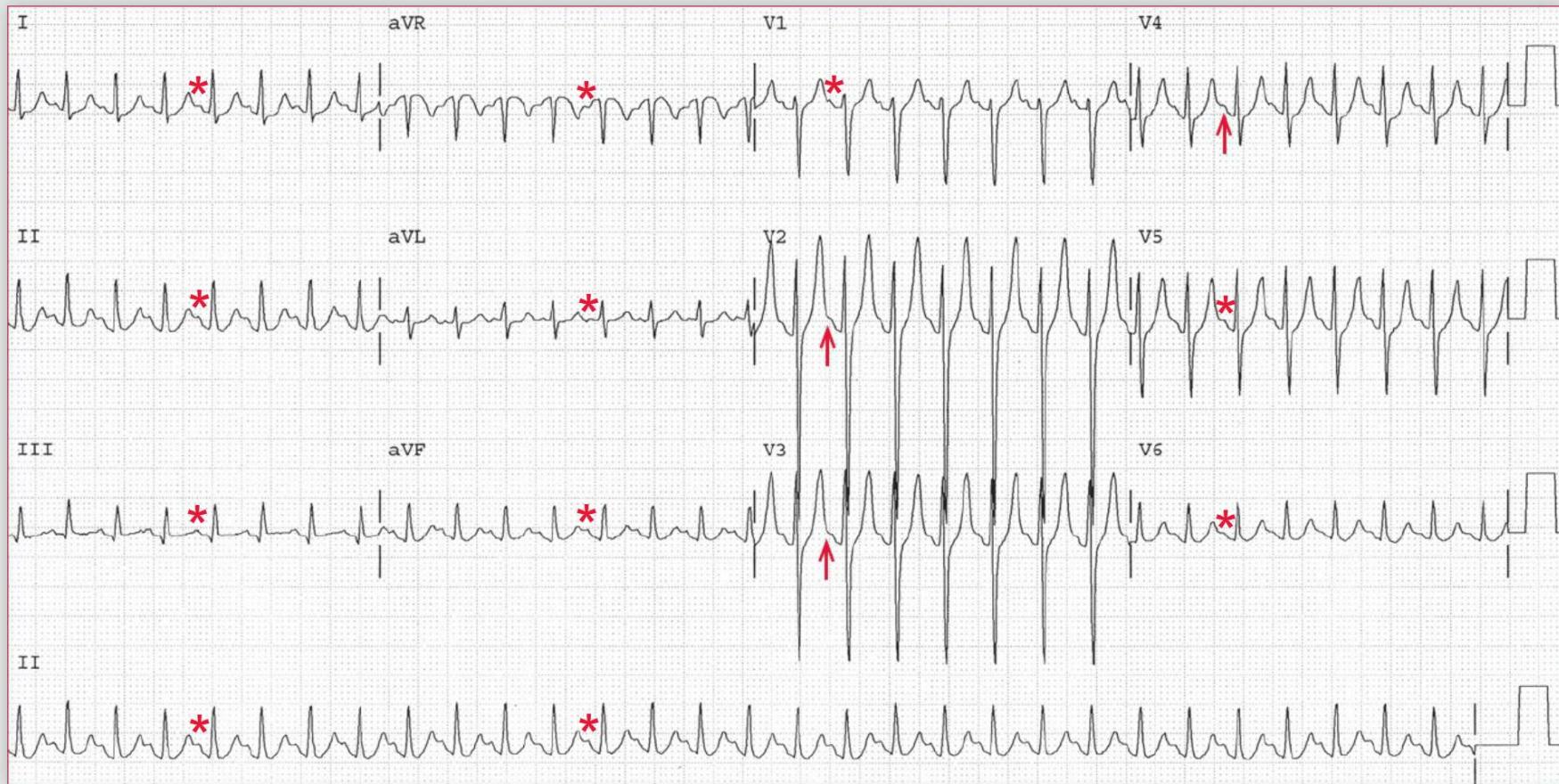
A 52-year-old man with no known cardiac history presents to the emergency department with fatigue and dizziness. He is hypotensive with a blood pressure of 74/50 mm Hg. You obtain an ECG.

Which of the following clinical scenarios does not match this patient's clinical presentation?

- A. Urosepsis**
- B. Intermittent torsade de pointes**
- C. Pulmonary embolism**
- D. Adrenal insufficiency**



Podrid's Real-World ECGs



ECG 3 Analysis: Sinus tachycardia

There is a regular rhythm at a rate of 180 bpm. Although not obvious in every lead, a P wave (*) can be seen before each QRS complex, particularly in leads I, II, III, and aVF. In the precordial leads, P waves (↑) can be seen at the very end of the T waves. The PR interval is constant (0.12 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Therefore, this is sinus tachycardia. In the presence of sinus tachycardia, the P waves are often superimposed on the T waves, especially if the PR interval is prolonged. Hence it is important to look carefully at the T waves when P waves are not readily apparent. It should be noted that T waves should be smooth in upstroke and downstroke. Notching or bumps on T waves are very suggestive of superimposed P waves.

The QRS complex duration (0.08 sec) and morphology are normal. The QRS axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (240/400 msec).

The PR interval is short, based on the definition of a normal PR interval (between 0.14 and 0.20 sec). However, the PR interval does change with heart rate as a result of changes in sympathetic and parasympathetic inputs into the AV node. Sinus tachycardia is generally the result of increased sympathetic activity, which causes an increase in conduction

velocity through the AV node. Therefore, sinus tachycardia is associated with a shortening of the PR interval. In contrast, sinus bradycardia, which is due to withdrawal of sympathetic stimulation and increased parasympathetic activity, is associated with a decreased conduction velocity through the AV node and hence an increase in the PR interval.

Sinus tachycardia is usually the result of sympathetic activation or an increase in circulating catecholamines. There are many possible etiologies for sinus tachycardia with hypotension, including any severe infection with or without sepsis, pulmonary embolism, adrenal insufficiency, acute bleeding or hypovolemia, and cardiogenic shock. Torsade de pointes is a form of polymorphic ventricular tachycardia that results from a long QT interval. Congenital long QT syndrome is due to a genetic abnormality that results in a myocardial channelopathy. Although torsade de pointes is generally provoked by tachycardia in patients with congenital QT prolongation, the QT/QTc interval in this case is normal. Drug-induced torsade de pointes is often bradycardic or pause-dependent; that is, it is most often observed with bradycardia because the QT interval prolongs further with slower heart rates. On this ECG, the QT/QTc interval is within the normal range and there is sinus tachycardia; hence torsade de pointes as the cause for this patient's symptoms is not likely. ■

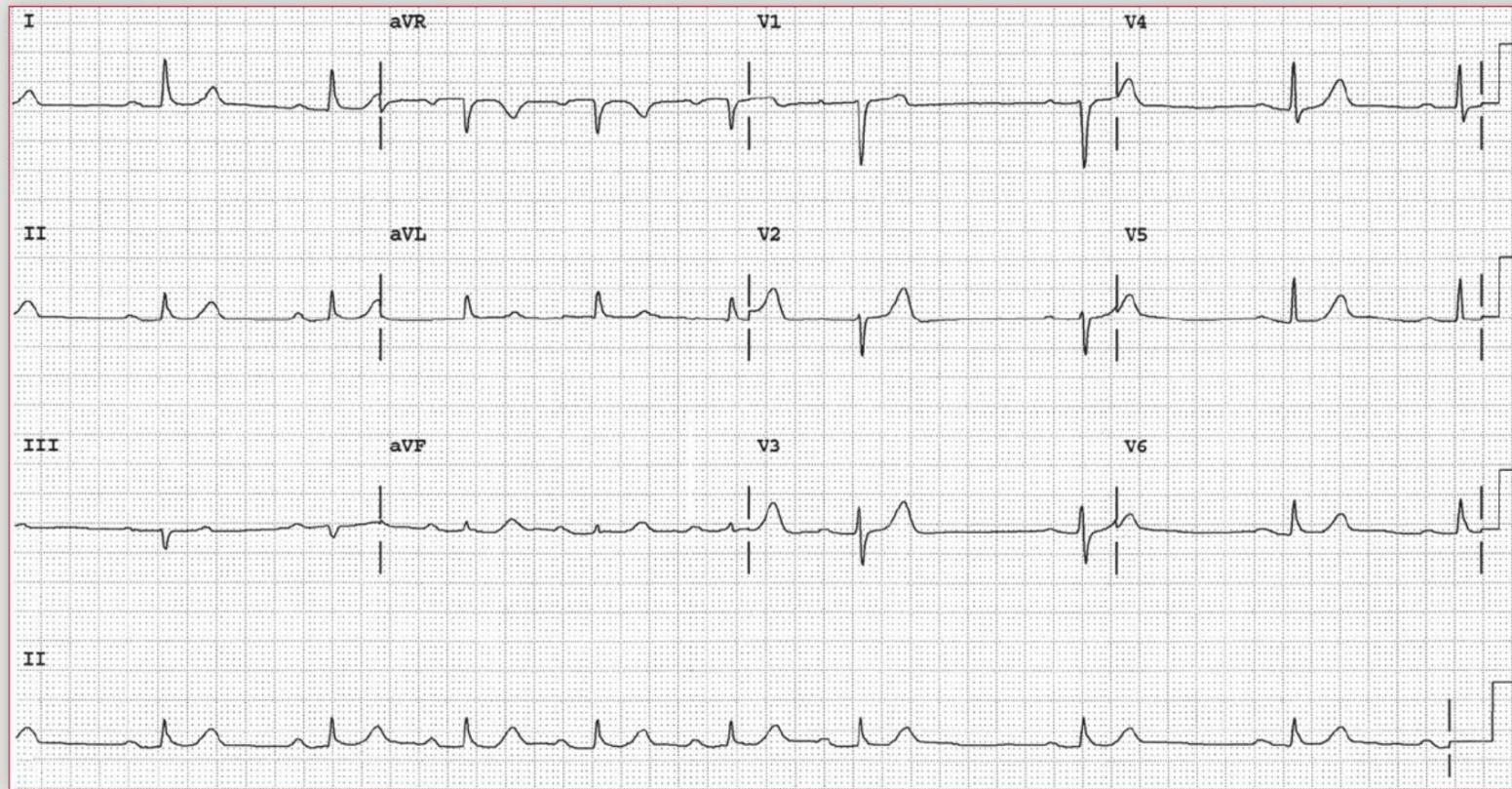
Notes

You and a colleague examine a 28-year-old patient together in your clinic. The patient is being seen for a routine annual visit and is asymptomatic. On physical examination, the patient is afebrile, with a blood pressure of 120/80 mm Hg and an irregular pulse. Head and neck examination and neurologic examination are unremarkable. You observe a biphasic jugular venous pressure (a and v wave present) at 6 cm without jugular venous distention. Carotid pulses have normal upstrokes. Lungs are clear on bilateral auscultation and percussion. Aside from an irregular heartbeat, the cardiac exam is unremarkable with no murmurs or rubs. The abdomen

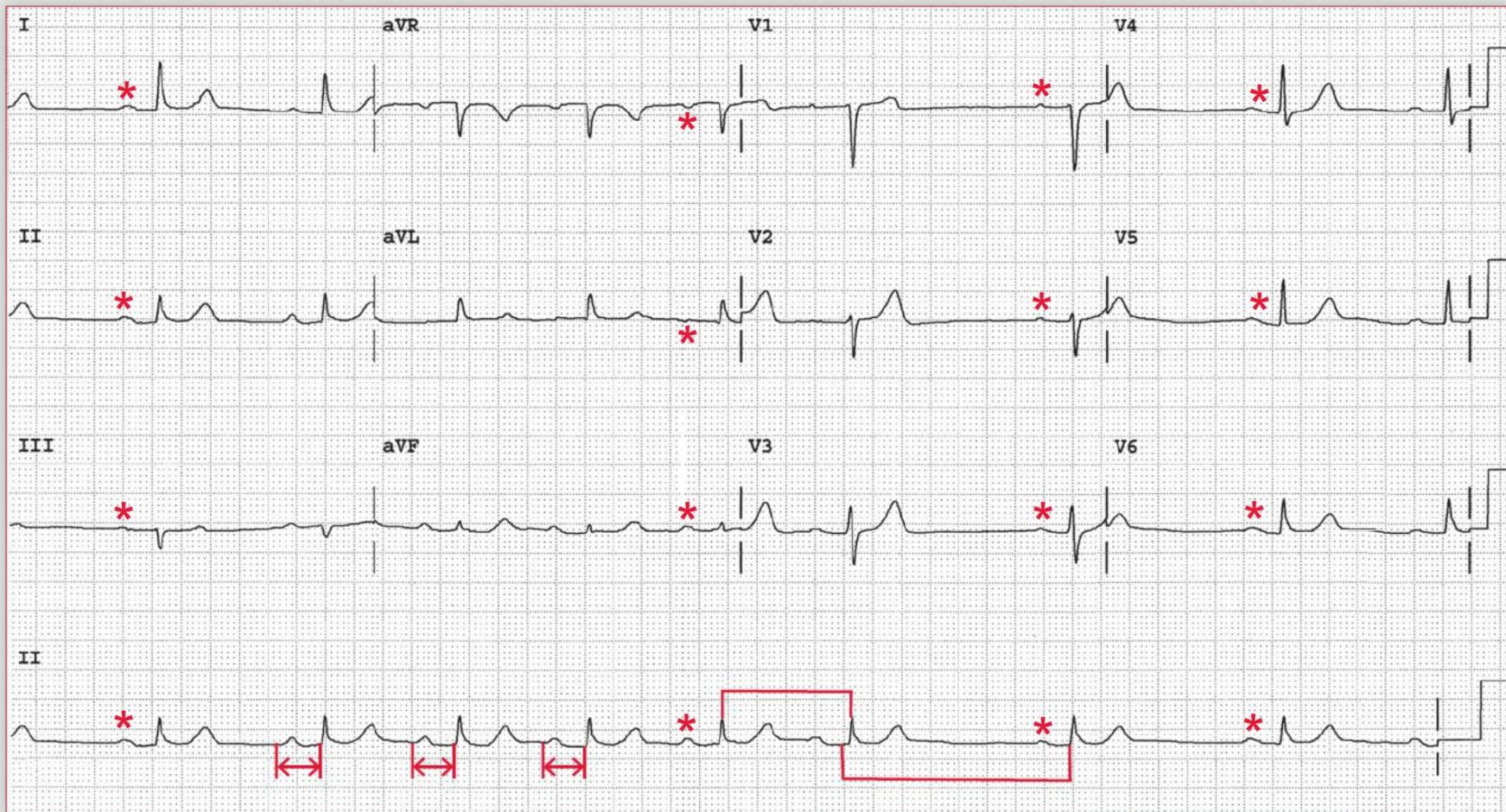
is soft and nontender, and the extremities are warm and well perfused. Your colleague says that the patient must be in atrial fibrillation given the irregular pulse, but you disagree with his conclusion. An ECG is obtained.

What is the underlying rhythm?

How did you know that the patient was not in atrial fibrillation?



Podrid's Real-World ECGs



ECG 4 Analysis: Sinus arrhythmia, first-degree AV block

The rhythm is irregularly irregular with a heart rate varying between 38 bpm (◻) and 68 bpm (◻). There is a P wave (*) before each QRS complex, and the P-wave morphology and PR interval (↔) are stable (0.28 sec). The P wave is positive in leads I, II, aVF, and V4-V6. This is, therefore, sinus arrhythmia and there is also first-degree AV block (prolonged AV conduction). Sinus arrhythmia is related to respiration (*ie*, it is a respirophasic arrhythmia). There are changes in sinus rate related to inspiration (heart rate increases) and expiration (heart rate decreases) that are mediated by neurocardiogenic reflexes.

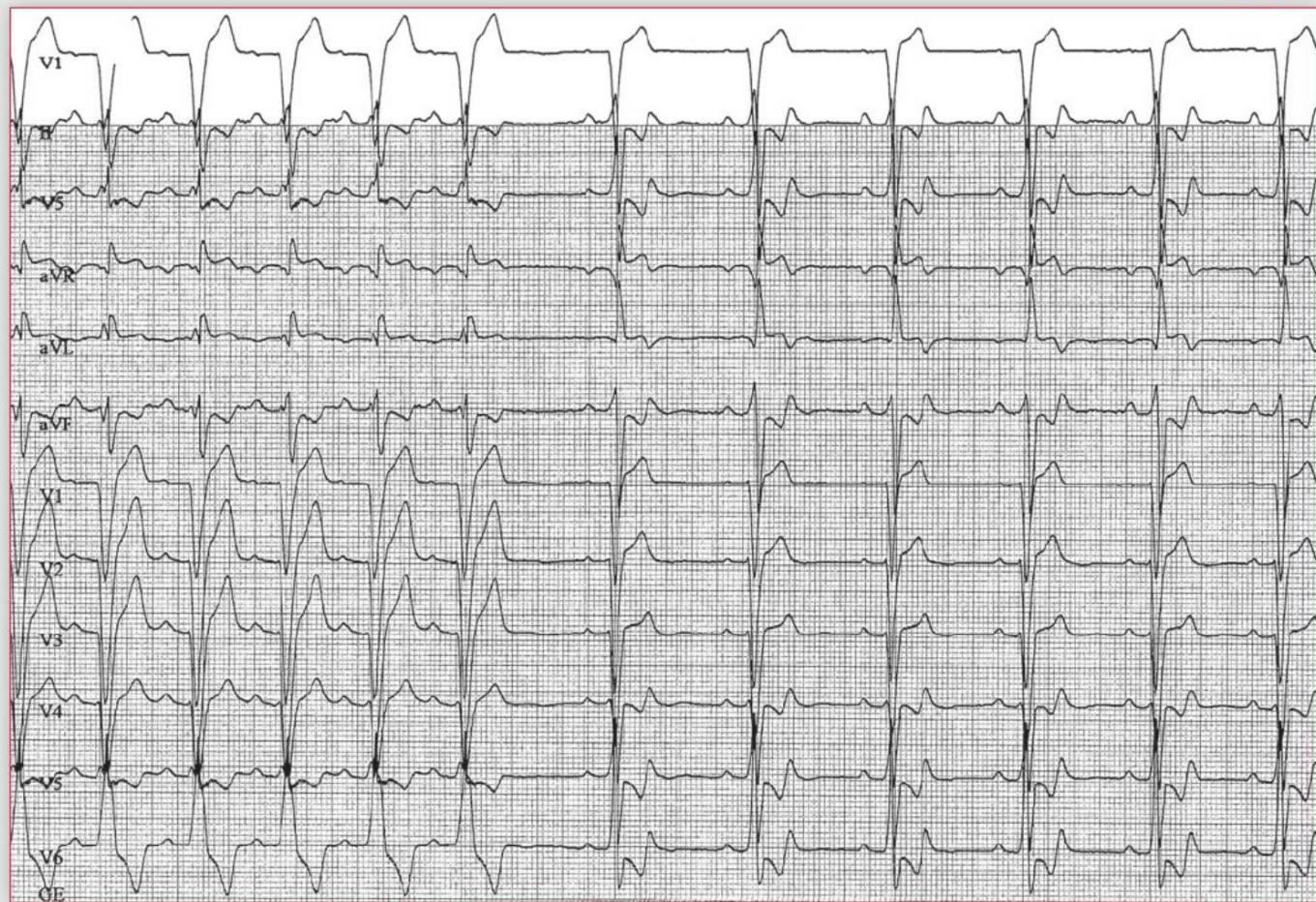
Upon inspiration, venous return to the heart increases due to negative intrathoracic pressure. An increase in venous return results in increased stretch of the myocardial fibers, which signals a decrease in parasympathetic activation of the vagus nerve as well as an increase in automaticity of pacemaker tissue due to a mechano-electrical feedback mechanism. Hence, an increase in venous return causes an increase in heart rate. With expiration, sympathetic activation decreases while parasympathetic activation increases and hence heart rate slows. The QRS complexes are normal in duration (0.08 sec) and morphology. The QRS axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/440 sec). The T waves are normal (asymmetric with a slower upstroke and more rapid downstroke).

Only three supraventricular rhythms are irregularly irregular: sinus arrhythmia, multifocal atrial tachycardia/wandering atrial pacemaker (or multifocal atrial rhythm), and atrial fibrillation. The presence of one P-wave morphology and a stable PR interval identifies sinus arrhythmia. The presence of three or more different P-wave morphologies (and PR intervals) without any one P wave being dominant is seen with wandering atrial pacemaker or multifocal atrial rhythm (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm). The hallmark of atrial fibrillation is the lack of discernible, organized P waves but the presence of rapid and irregular fibrillatory waves. The absence of an organized P wave results in the loss of organized atrial contraction. Recall that the jugular venous waveform reflects the right atrial pressure tracing and is biphasic due to an a wave (atrial contraction wave) and a v wave (venous return wave). When an individual is in atrial fibrillation, examination demonstrates that the jugular venous waveform is no longer biphasic and becomes monophasic with only a v wave and a loss of the a wave. Since the patient in this scenario had biphasic jugular venous pressure, the patient could not be in atrial fibrillation. ■

Core Case 5

A 55-year-old woman with no known cardiac disease presents with intermittent palpitations that have never been associated with dizziness,

ECG 5A



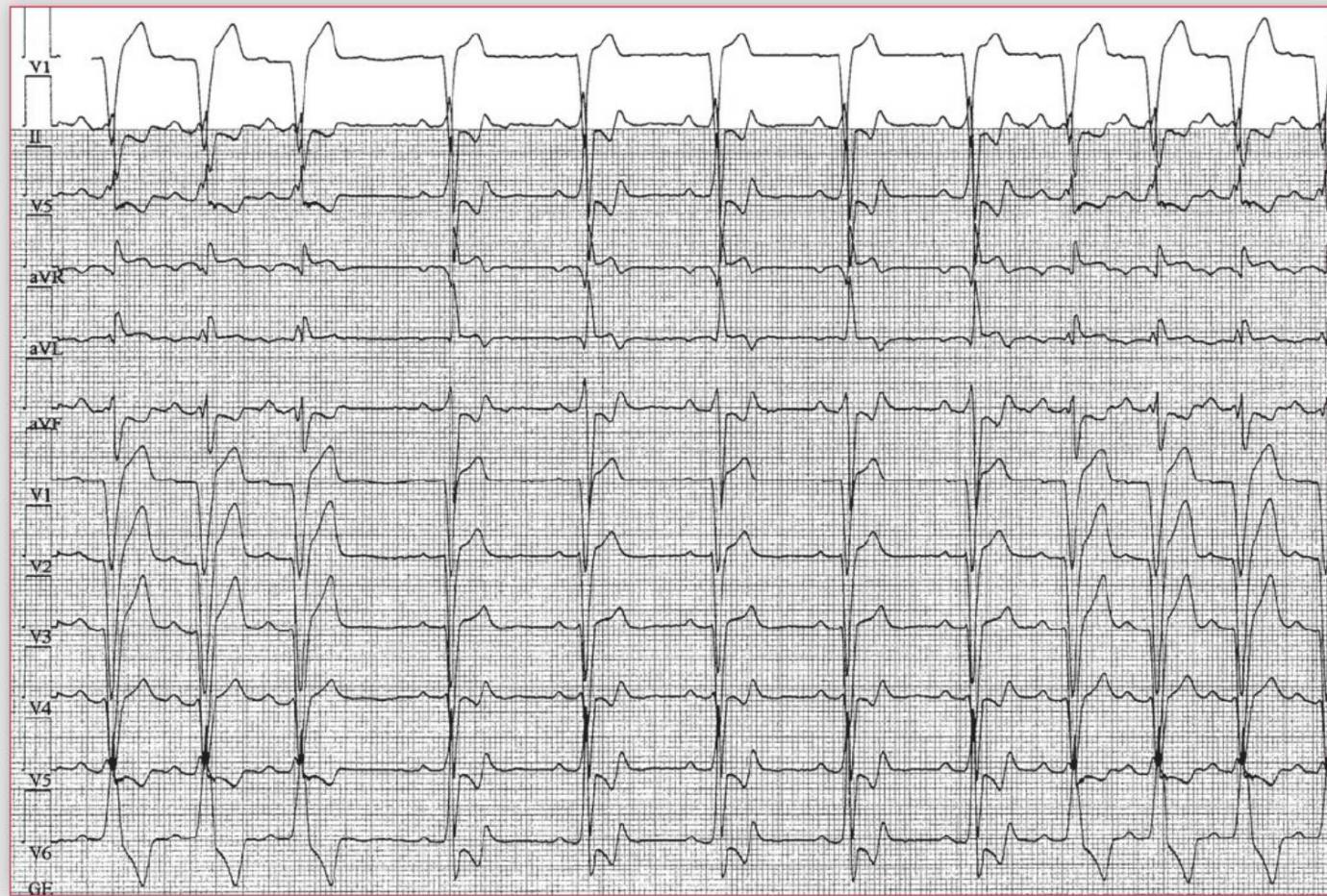
lightheadedness, or syncope. You obtain an ECG while she is symptomatic (ECG 5A). Several minutes later a second ECG is obtained (ECG 5B).

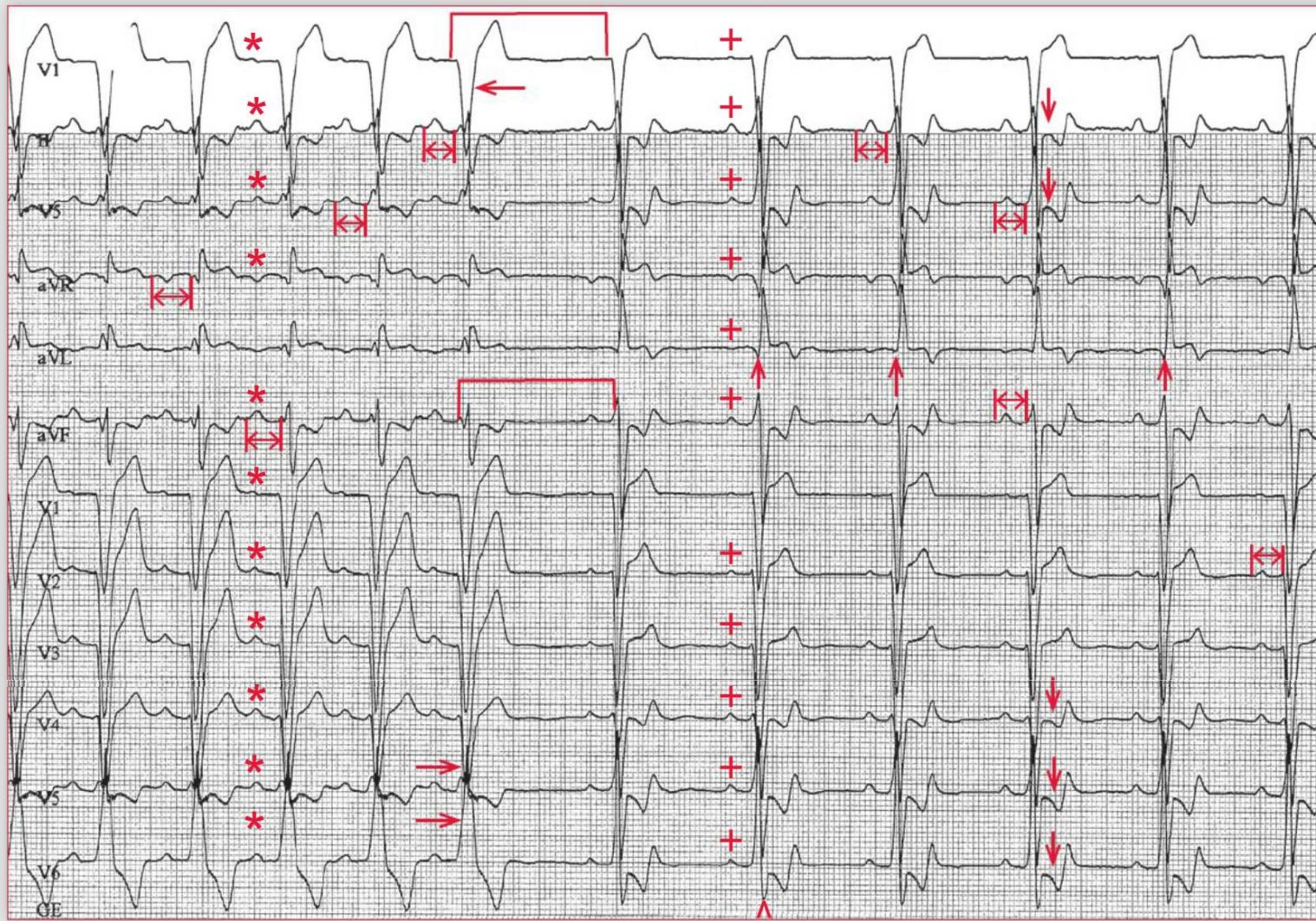
What is the etiology of her palpitations?

How do you explain the changes in QRS morphology on the ECG?

If this patient needs a stress test, what type of test should be obtained?

ECG 5B





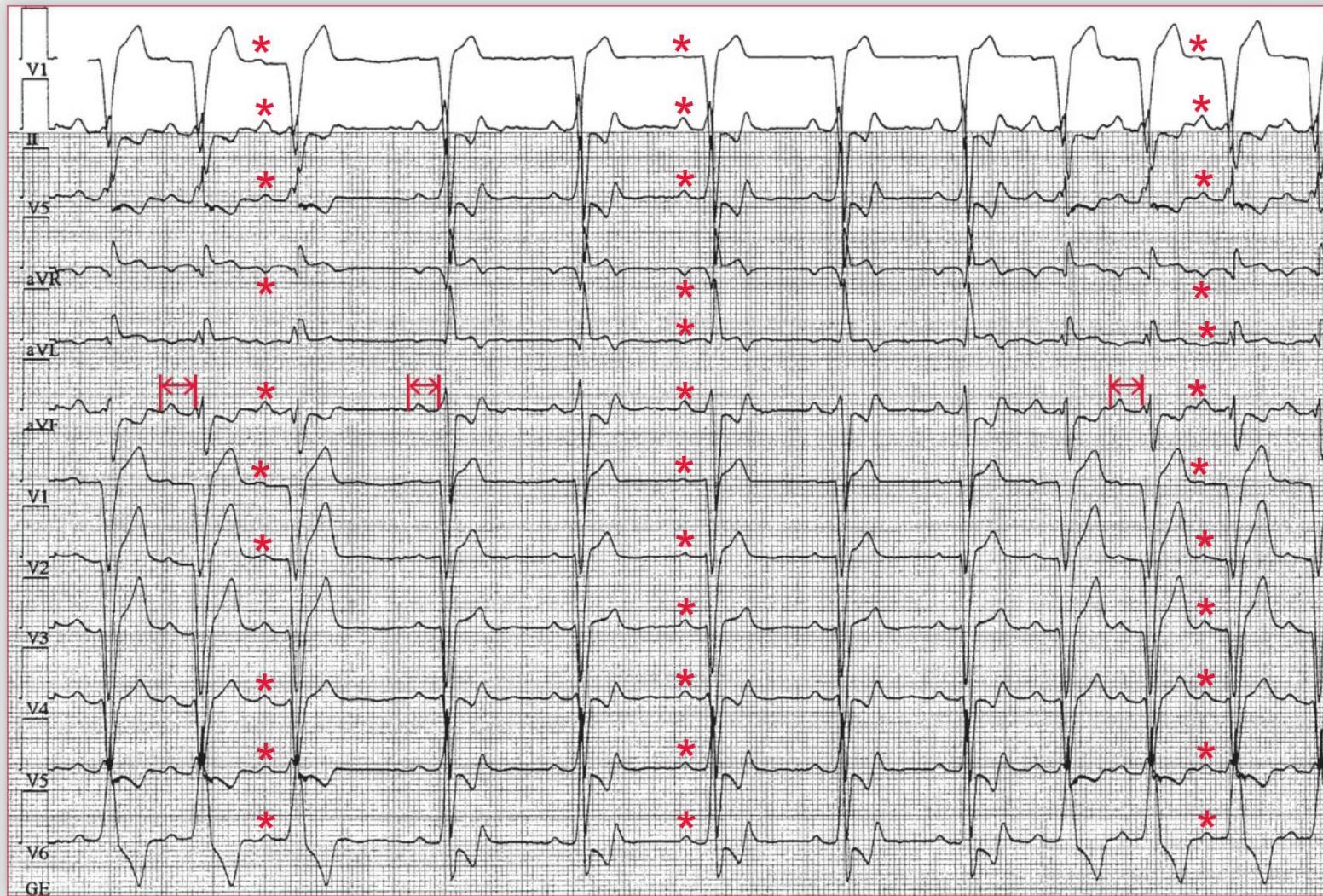
ECG 5A Analysis: Sinus nodal reentrant tachycardia alternating with normal sinus rhythm, intraventricular conduction delay, rate-related left bundle branch block

The initial part of ECG 5A shows a regular rhythm at a rate of 94 bpm. There is a P wave (*) in front of each QRS complex with a stable PR interval (0.18 sec) (↔). The P waves are positive in leads II, aVF, and V4-V6. Hence this is a sinus rhythm. The QRS complexes are wide (0.16 sec) and have a left bundle branch block (LBBB) morphology (QS complex in lead V1 [←] and broad R wave in leads V5-V6 [→]). There is an abrupt slowing of the rate to 56 bpm (◻). At the slower rate a P wave (+) is seen before each QRS complex and the P wave has the same morphology, axis, and PR interval (↔) as were seen when the rate was 94 bpm. Hence both rhythms are sinus. This is characteristic of sinus node reentry, which has an abrupt slowing of the sinus rate rather than a gradual slowing as would be seen with sinus tachycardia. It should be noted that when the rate is slower, the QRS complex duration is narrower (0.10 sec), although the morphology of the QRS complex is similar to that seen at the higher rate. However, as the QRS complex width is less than 0.12 second, this is an intraventricular conduction delay (IVCD) and not a full LBBB. (IVCD with a morphology resembling an LBBB is often referred to as incomplete LBBB.) Unlike the LBBB complex, the narrower QRS complex has a small Q wave (septal Q wave) in lead aVL (↑), which further confirms that this is no longer an LBBB. Septal forces are not seen with LBBB as the septal or medial fascicle that innervates the septum originates from the left bundle.

Additionally, the narrower QRS complex has a terminal S wave in lead V6 (Λ), indicating terminal forces going from left to right. This is not seen with an LBBB as all of the forces are directed right to left. The occurrence of the wide QRS complex with the faster rate indicates that the LBBB is rate related. Also noted are significant ST-T wave abnormalities (↓) in leads II and V4-V6. The QT/QTc intervals are prolonged (420/520 msec) but normal when considering the prolonged QRS complex duration (340/430 msec).

At a slower heart rate there is an IVCD, which does not preclude evaluation of ST-segment changes with exercise. However, the development of a complete LBBB at faster heart rates (*ie*, rate-related LBBB) would limit the usefulness of the ECG during an exercise test because ST-segment changes (or indeed any left ventricular abnormality) cannot be evaluated and cannot be used to diagnose ischemia with an LBBB. With an LBBB, left ventricular activation is no longer via the normal His-Purkinje system but rather occurs by direct myocardial activation via the right bundle and right ventricle. Hence an ECG stress test alone would not be able to detect the presence of ischemia. Therefore, if this patient were to undergo stress testing, she would need additional assessment with some type of imaging, either nuclear imaging (*ie*, sestamibi) or echocardiography.

continues



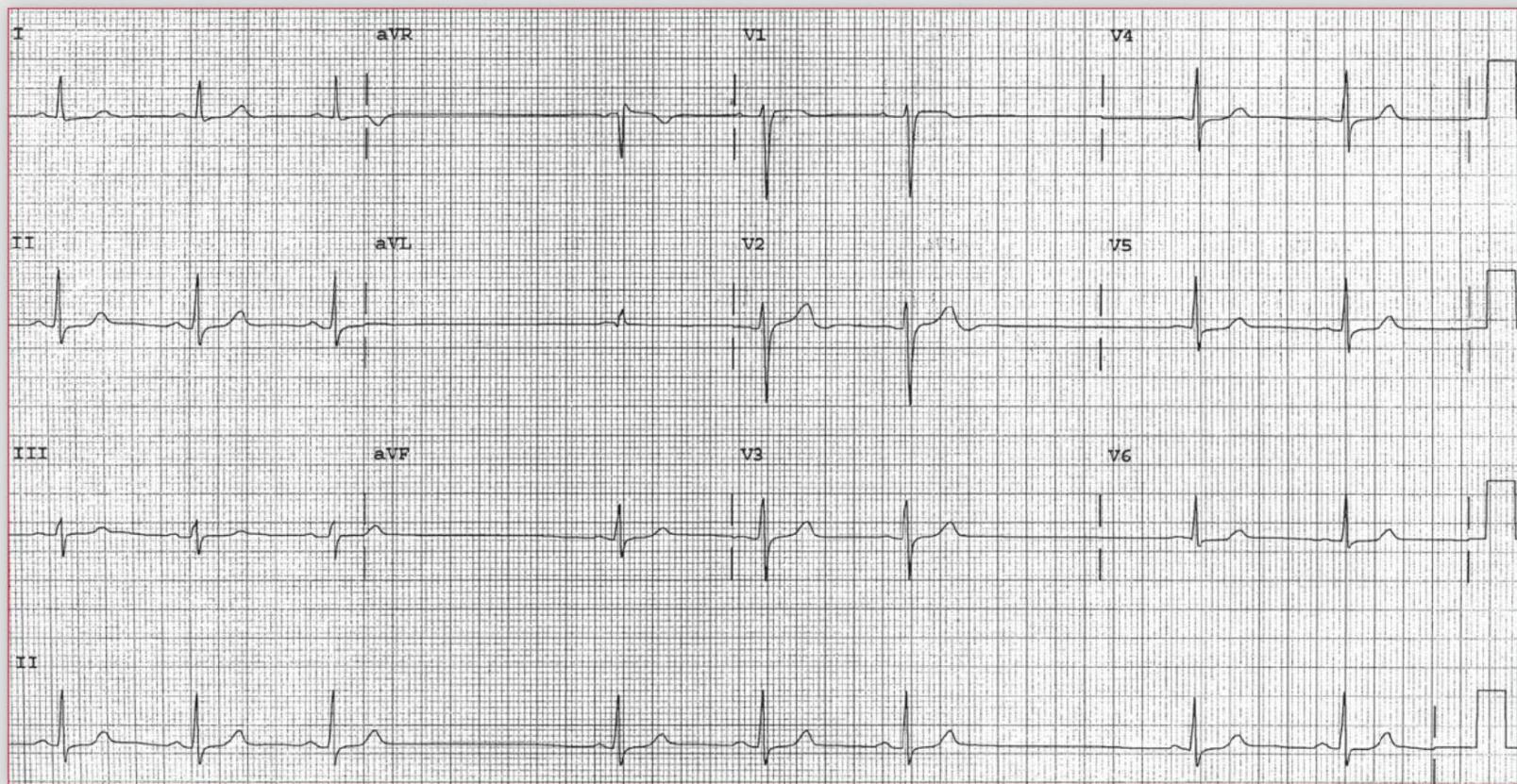
ECG 5B Analysis: Sinus nodal reentrant tachycardia alternating with normal sinus rhythm, intraventricular conduction delay, rate-related left bundle branch block

ECG 5B shows an initial rate of 94 bpm with an abrupt slowing to 58 bpm. The rate then abruptly returns to 94 bpm. The same P wave (*) and PR interval (↔) are present before each QRS complex, regardless of the rate. The P wave and PR interval are identical to those seen in ECG 5A. This is characteristic of sinus nodal reentrant tachycardia (*ie*, an abrupt onset and offset of the elevated heart rate). The QRS complex morphology and QT/QTc intervals are identical to what is seen in ECG 5A. Noted are an IVCD as well as a rate-related LBBB, as were seen in ECG 5A. ■

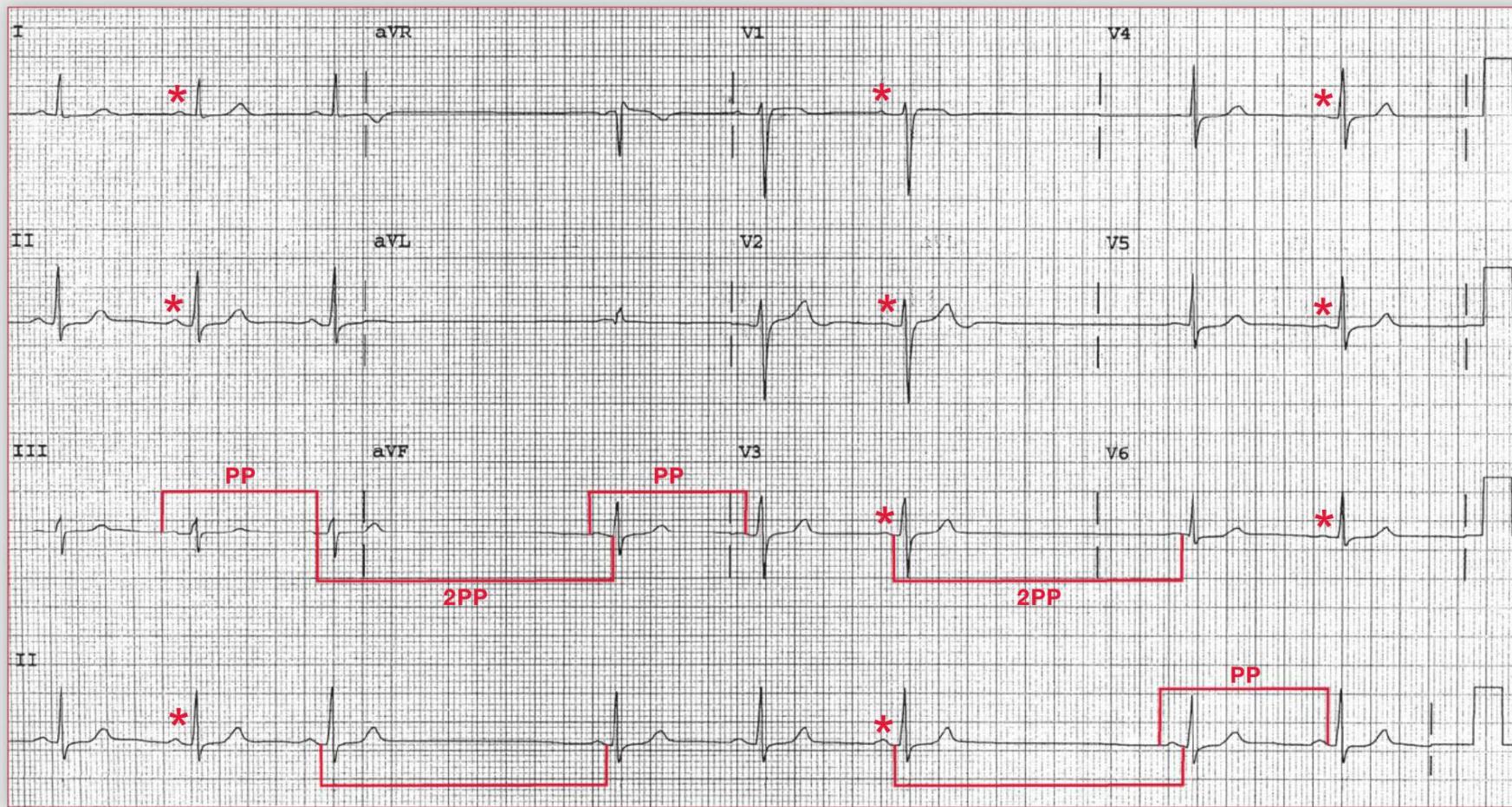
Notes

A 62-year-old man with an extensive history of smoking and a recent diagnosis of squamous cell carcinoma of the neck that was treated with radiation now presents with 2 weeks of intermittent dizzy spells and one episode of syncope. You obtain the following ECG while performing carotid massage, which provokes the patient's symptoms.

What is the rhythm abnormality?
What is the clinical diagnosis?
How would you manage this patient?



Podrid's Real-World ECGs



ECG 6 Analysis: Normal sinus rhythm, sinus node exit block

The rhythm is primarily regular at a rate of 60 bpm. There are P waves (*) before each QRS complex with a stable PR interval (0.16 sec). There are two long RR (or PP) intervals without any P wave during the intervals (□). These represent two sinus pauses, the duration of which is equal to two sinus intervals (■) (based on measurement of the PP intervals of the two sinus complexes immediately before and after the pauses). Therefore, this represents sinus node exit block. With sinus node exit block, the sinus impulses are regular but there is a failure of one sinus impulse to exit the sinus node area to activate the atria; hence no P wave is present. Since the sinus node impulses are regular, the PP interval around the pauses is equal to two sinus intervals.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec).

The slowing of the sinus rate due to sinus node exit block with light pressure on the carotid artery is a manifestation of carotid sinus

hypersensitivity (CSH). CSH is an exaggerated carotid baroreceptor response that results in dizziness or syncope from bradycardia (cardio-inhibitory response) or peripheral vasodilation (vasodepressor response). Bradycardia can be due to sinus node and/or AV node dysfunction. Increased transmural pressure of the carotid sinus creates afferent impulses to the nuclei tractus solitarius in the brain stem, resulting in efferent impulses via the sympathetic and vagus nerves that affect heart rate and vasomotor tone. CSH is typically seen in elderly patients and is associated with underlying neck pathology such as tumors, post-surgical scarring, or radiation fibrosis.

The reproduction of symptoms and bradycardia with carotid sinus massage confirms the diagnosis of CSH. Management includes stopping all nodal suppressive agents and maintaining adequate intravascular volume. Persistence of these symptoms in a patient with syncope due to cardio-inhibitory CSH is a class I indication for permanent pacemaker placement. ■

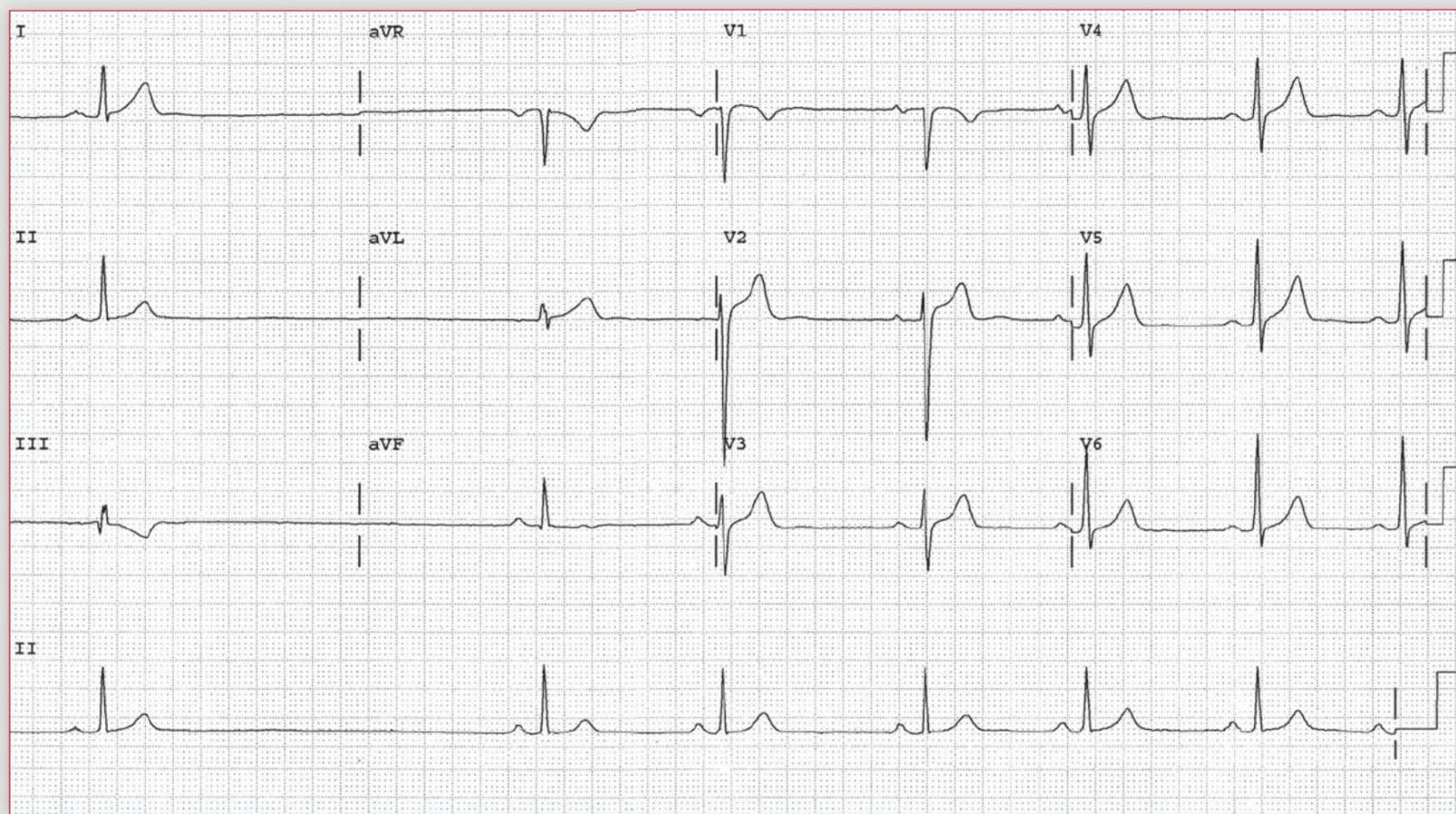
Notes

A 76-year-old man with no previous cardiac history presents following 2 weeks of intermittent lightheadedness and pre-syncope. He does have a history of hypertension treated with an angiotensin-converting enzyme inhibitor. While feeling a pulse, occasional long pauses are appreciated. An ECG is obtained.

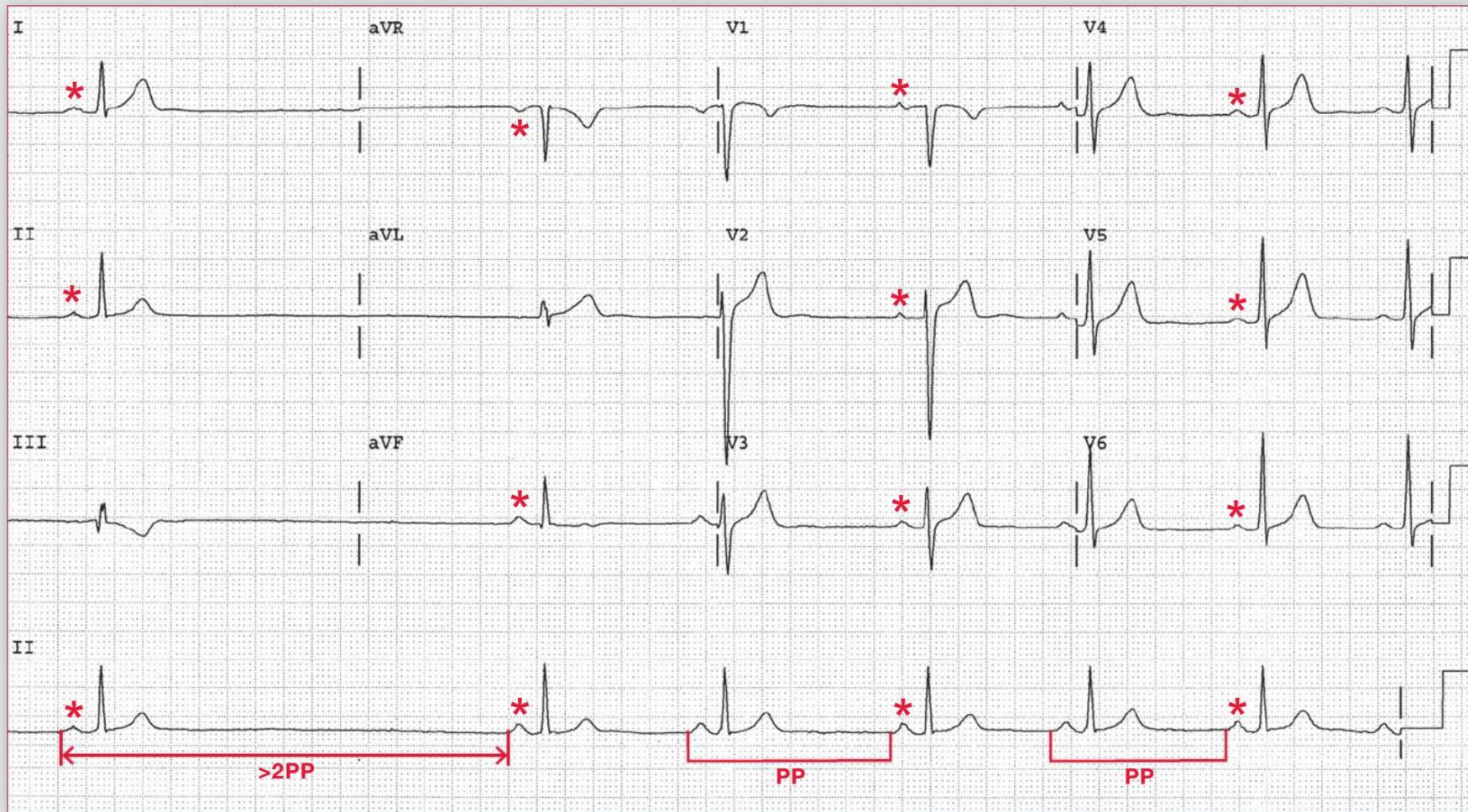
What abnormality is notable on the ECG?

Does this abnormality suggest a diagnosis for this patient?

What therapy might be indicated?



Podrid's Real-World ECGs



ECG 7 Analysis: Sinus bradycardia, sinus arrhythmia, sinus node arrest

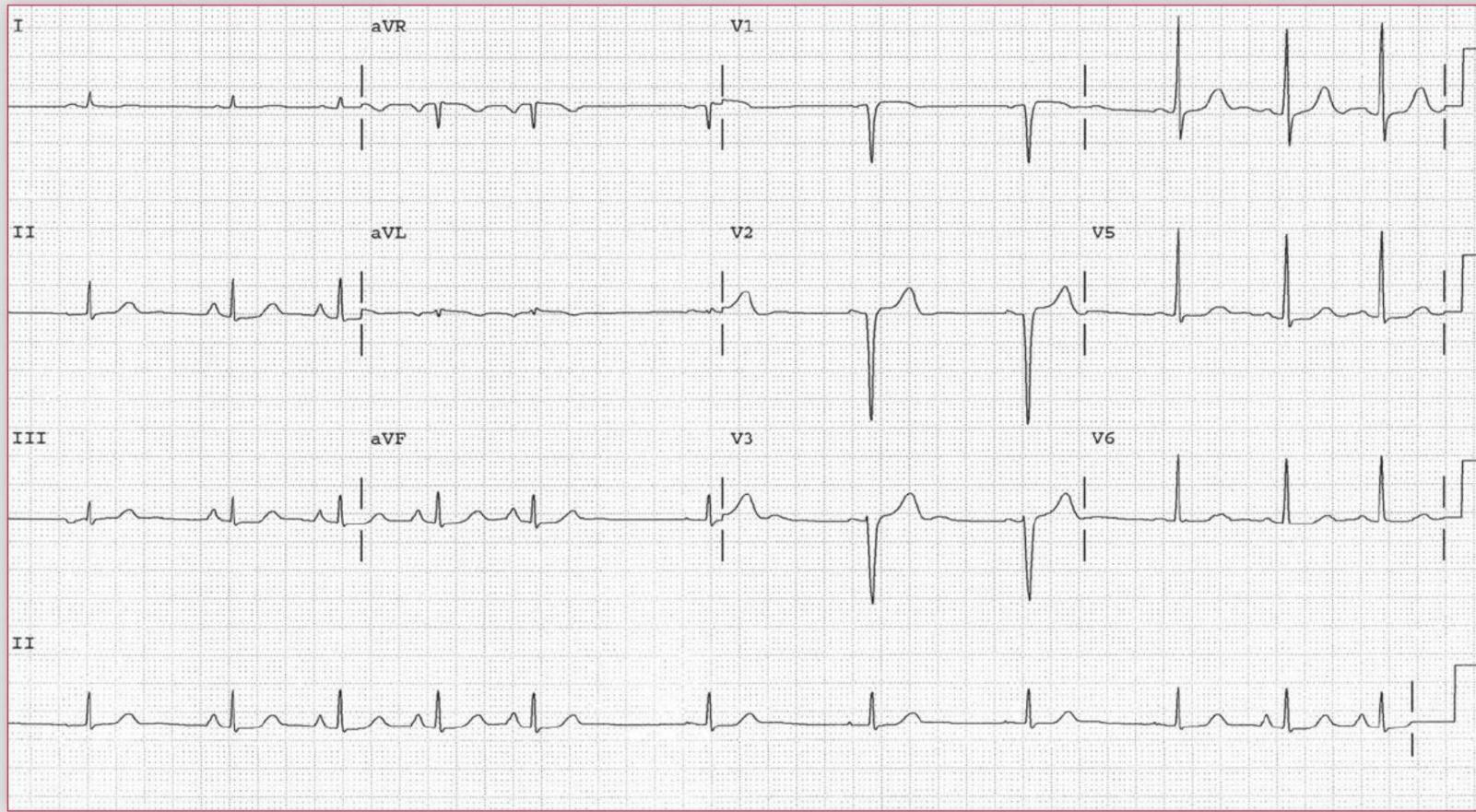
There is sinus bradycardia with a rhythm that is irregularly irregular and a heart rate that varies between 44 and 54 bpm. There is a P wave before each QRS complex (*) with a stable PR interval (0.22 sec). The P-wave morphology is constant, and it is positive in leads I, II, aVF, and V4-V6. Hence this is sinus bradycardia with sinus arrhythmia also present. Sinus arrhythmia presents with an irregularly irregular rhythm with a P wave of constant morphology before each QRS complex and a stable PR interval. Noted is a very long RR interval (↔); during this long pause there is no P wave present. Therefore, this is a sinus pause. The PP interval around the pause is longer than two PP intervals (∟) (even when the sinus arrhythmia is considered), and hence this represents sinus node arrest. In this situation, the sinus node fails to generate an impulse and then recovers after a variable period of time.

The presence of sinus bradycardia and sinus arrhythmia may be the result of drugs that suppress sinus node automaticity, such as digoxin, a β -blocker, or a calcium-channel blocker. However, this patient is not taking any of these medications. Other possible causes include high vagal tone and intrinsic disease of the sinus node. The presence of sinus node arrest with a long PP interval suggests that there is underlying sinus node dysfunction, which might be termed sick sinus syndrome. Evidence of sinus node dysfunction or sick sinus syndrome in a patient with symptoms that are likely the result of bradycardia is an indication for insertion of a permanent pacemaker. ■

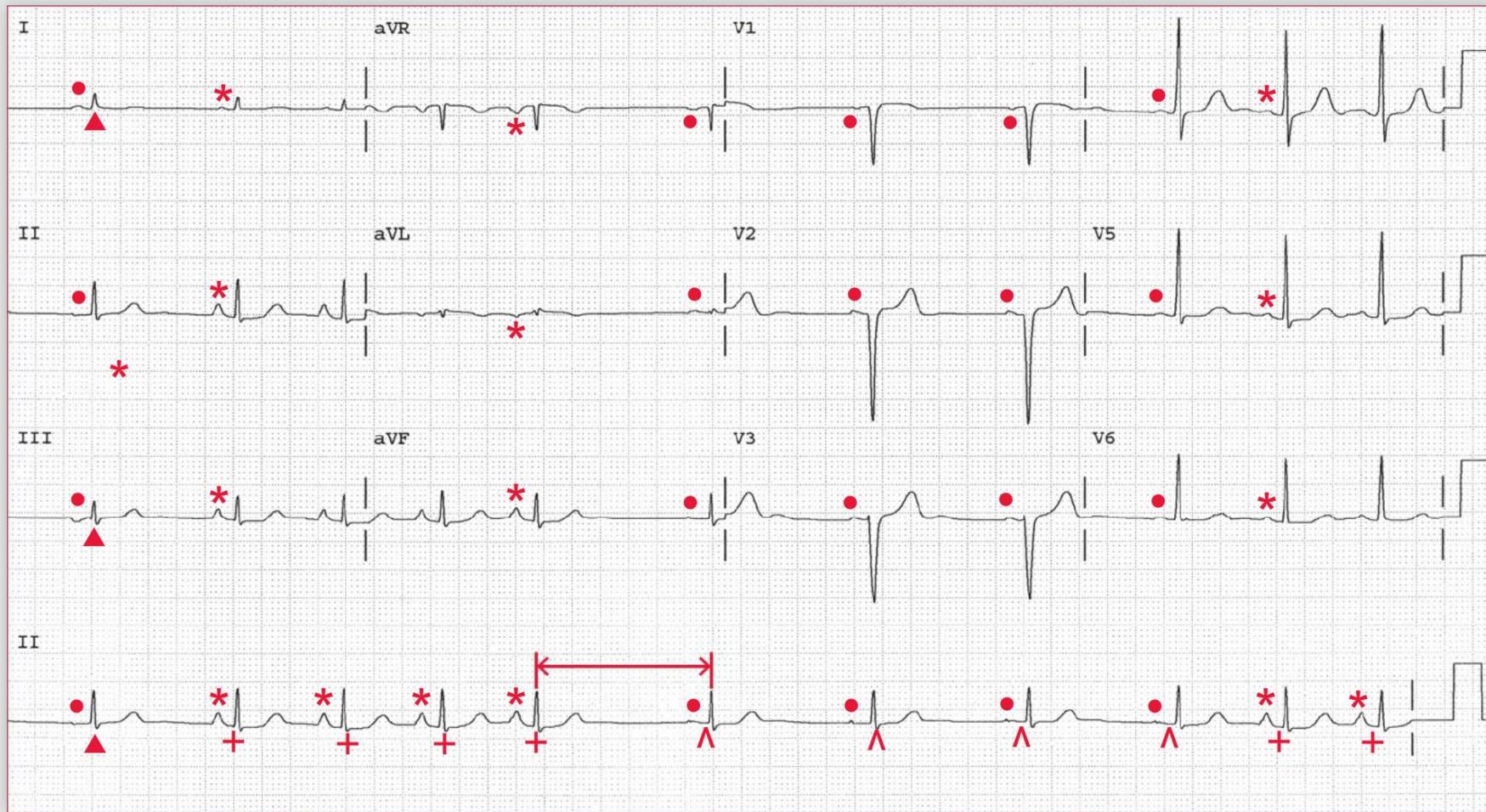
Notes

A patient who underwent an appendectomy yesterday has the following ECG and is started on a β -blocker for rate control of postoperative atrial fibrillation. A cardiology consultation is obtained.

What is the rhythm diagnosis?
Should the patient remain on a β -blocker?



Podrid's Real-World ECGs



ECG 8 Analysis: Normal sinus rhythm, sinus node arrest with escape atrial rhythm

At first glance, one might think this is underlying sinus bradycardia with a faster ectopic atrial rhythm. On closer inspection, however, the faster rate represents sinus rhythm. The second, third, fourth, and fifth QRS complexes (+) as well as the last two complexes (+) have a constant rate of 90 bpm. There is a P wave (*) with a constant morphology before each of these QRS complexes, and there is a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6, representing normal sinus rhythm. Following the fifth QRS complex there is a pause (↔), after which there are four QRS complexes (Λ) that have the same morphology as all the other QRS complexes. These QRS complexes are also preceded by P waves (●), but the P-wave morphology is different than that of the sinus complexes (second through fifth complexes). The morphology in leads II and aVF is atypical for a sinus P wave as the waveform is small and very narrow. In addition, the PR interval is slightly longer (0.18 sec) than the PR interval of the sinus complexes. These are, therefore, atrial complexes. There is a regular RR interval at a rate of 60 bpm. This is a manifestation of sinus node arrest with an escape atrial rhythm, after which a normal sinus rhythm recurs. Indeed, the first QRS complex (▲) is preceded by the same abnormal P wave (●) and is also an atrial complex.

All the QRS complexes are the same, and the QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/470 msec).

Sinus node arrest may be the result of enhanced vagal tone, medications that affect sinus node activity (*ie*, β -blockers, digoxin, or calcium-channel blockers), or sinus node dysfunction (or possible sick sinus syndrome). In this patient there is an escape atrial focus that takes over pacemaker function in the absence of sinus node activity. As a result, asystole or severe bradycardia does not occur and the patient is without symptoms. The etiology for the sinus node arrest is unclear, as any of the above-mentioned etiologies may be implicated. The most likely etiology is β -blocker therapy. As the patient is no longer in atrial fibrillation, discontinuation of this medication should be considered.

There are no associated symptoms, so no other specific therapy is warranted. Although there is sinus node arrest, there is also an intact escape mechanism so bradycardia is not present. The presence of symptomatic bradycardia that persisted after the discontinuation of a β -blocker would be an indication for a pacemaker. ■

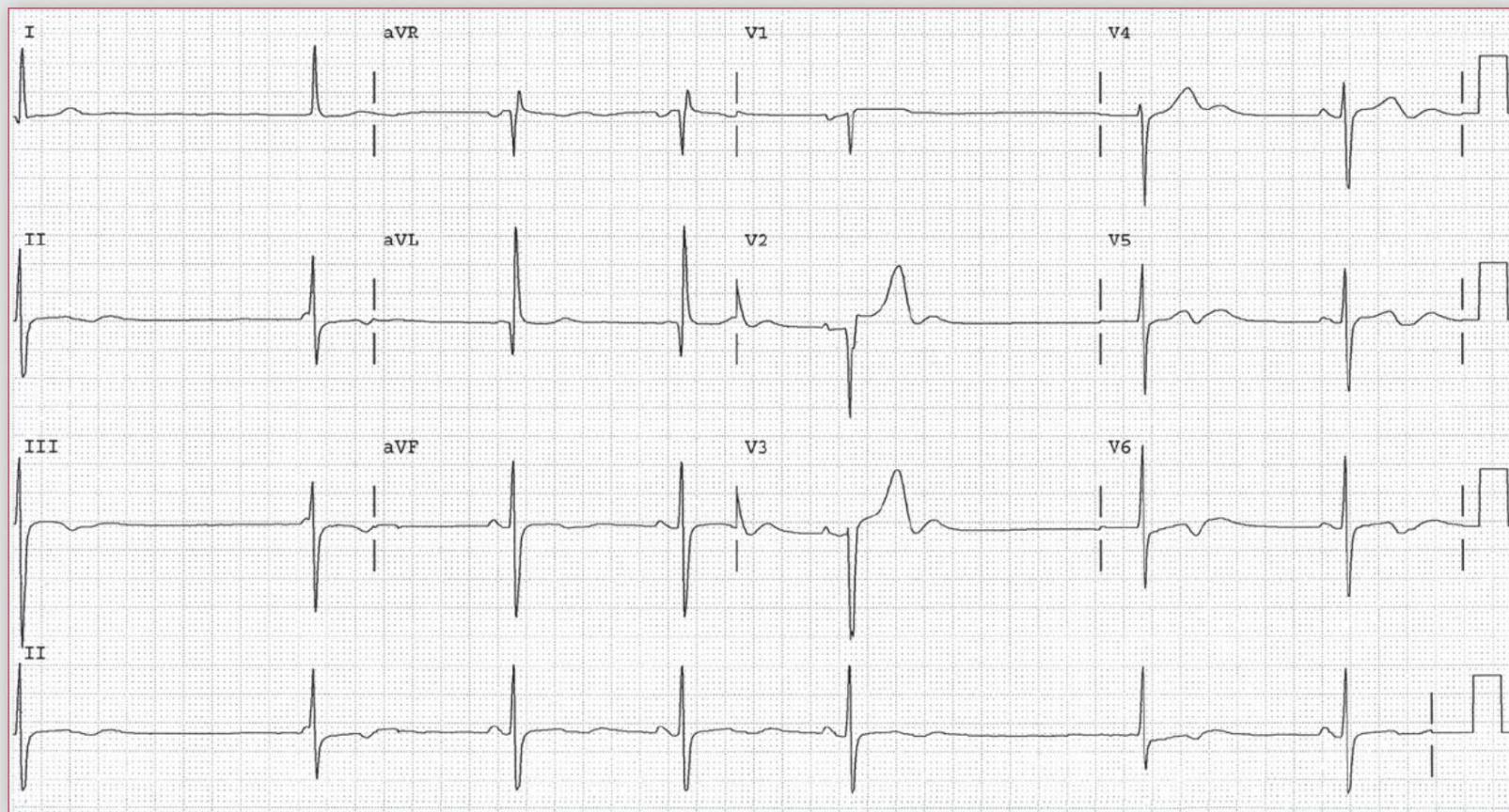
Notes

An 82-year-old otherwise healthy woman with hypertension treated with atenolol presents with viral gastroenteritis. She has had repeated bouts of vomiting and her oral intake has been severely limited over the past 3 days, but she was able to take her medication earlier today. The patient reports associated intermittent dizzy spells. You obtain the following ECG while she is having an episode of dizziness.

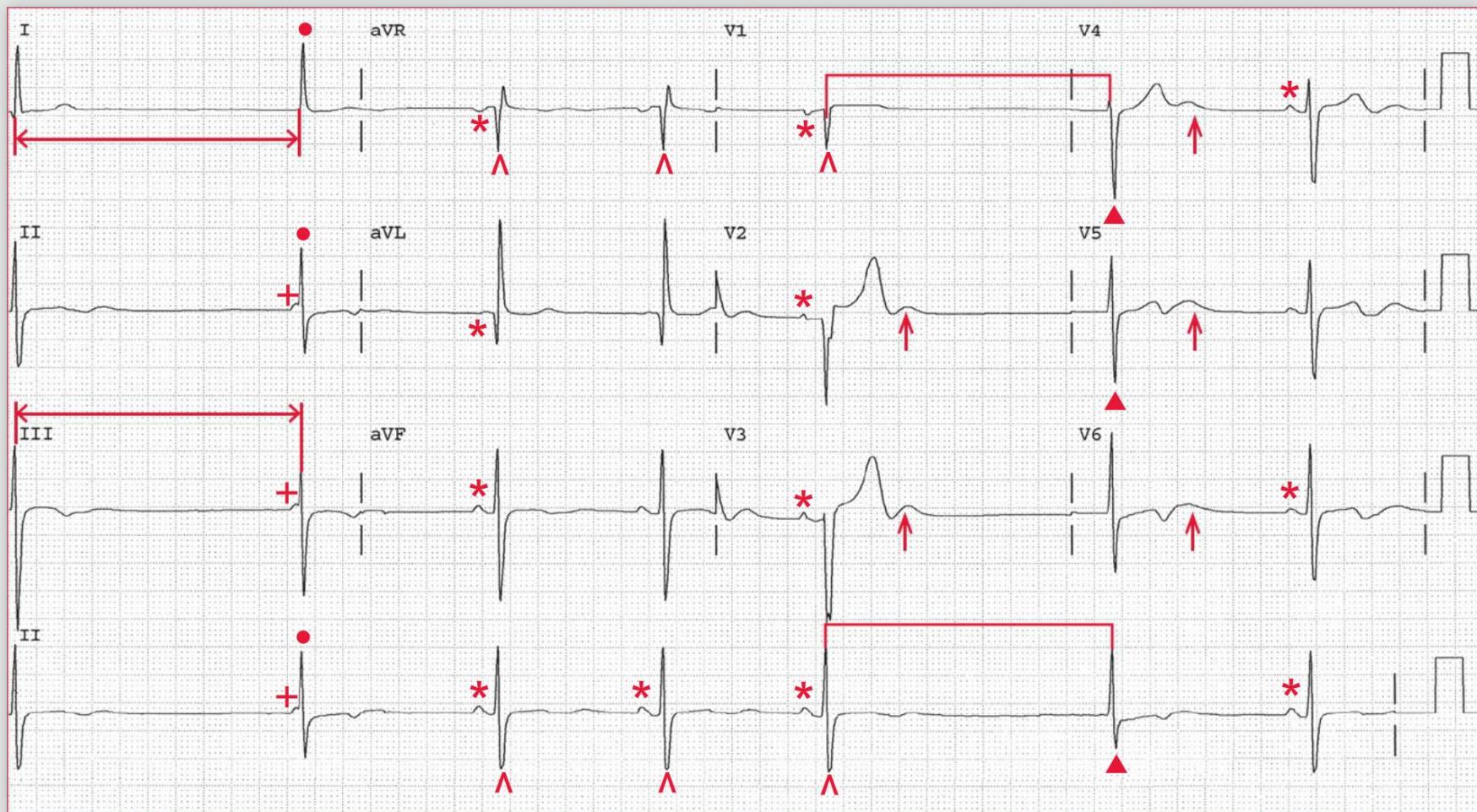
What is the rhythm disturbance?

What is the most likely etiology for this disturbance?

Where is the origin of the abnormal QRS complexes?



Podrid's Real-World ECGs



ECG 9 Analysis: Sinus bradycardia, sinus node arrest with junctional escape complex, U wave

The ECG shows an initial long RR interval (↔) during which there is no P wave. The second QRS complex (●) has a nonconducted P wave (+) that is superimposed on the upstroke of the R wave. The QRS complex is narrow (0.08 sec) with a normal morphology. It has the same morphology as the three subsequent QRS complexes (Λ), which are preceded by a P wave (*) with a stable PR interval (0.18 sec). The second QRS complex (●) is, therefore, an escape junctional complex. The P waves are positive in leads II, aVF, and V4-V6. Hence there is an underlying sinus rhythm. QRS complexes three to five (Λ) occur at a rate of 50 bpm and are sinus complexes. After the fifth QRS complex there is a long RR interval (□) that is ended by a narrow QRS complex (▲) that does not have a preceding P wave. This is another escape junctional complex. The duration of the pause is greater than two sinus PP intervals. Hence the rhythm is sinus bradycardia with two episodes of sinus node arrest with escape junctional beats at a rate of 30 bpm.

The QRS axis in the frontal plane is physiologically leftward, between 0° and -30° (positive QRS complex in leads I and II but negative QRS complex in lead aVF). The QT/QTc intervals are normal (480/440 msec). Following the T wave in leads V2-V6 is a prominent

U wave (↑). The U wave is believed to represent late repolarization, possibly of the His-Purkinje system, and is often seen in the right precordial leads. When prominent, tall U waves are seen through the entire precordium, hypokalemia is suggested.

Junctional escape rhythms exhibit QRS complexes that are similar in morphology to normally conducted QRS complexes (since they originate from the AV node or junction and are conducted to the ventricles via the normal His-Purkinje system), whereas ventricular escape rhythms will have wider QRS complexes that are abnormal in morphology and do not resemble a typical right or left bundle branch block (as myocardial activation is no longer via the normal His-Purkinje system but rather by direct myocardial activation) and typically occur at a slower rate.

Considering the patient's poor oral intake and repeated episodes of emesis, she is dehydrated and possibly has developed both hypokalemia and prerenal azotemia. Given that atenolol is predominantly cleared renally, the sinus node arrest is probably due to an increase in serum atenolol concentrations. ■

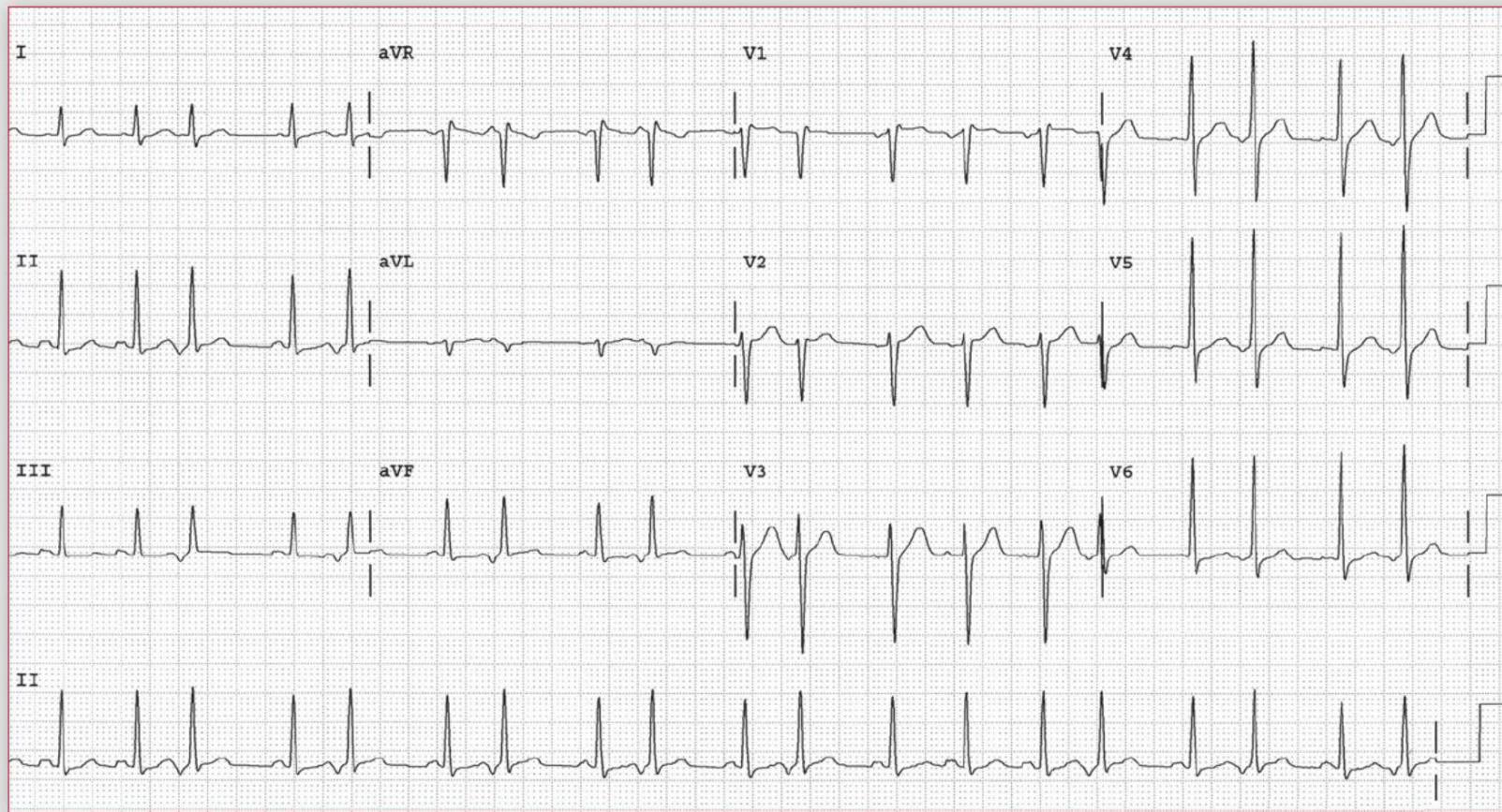
Notes

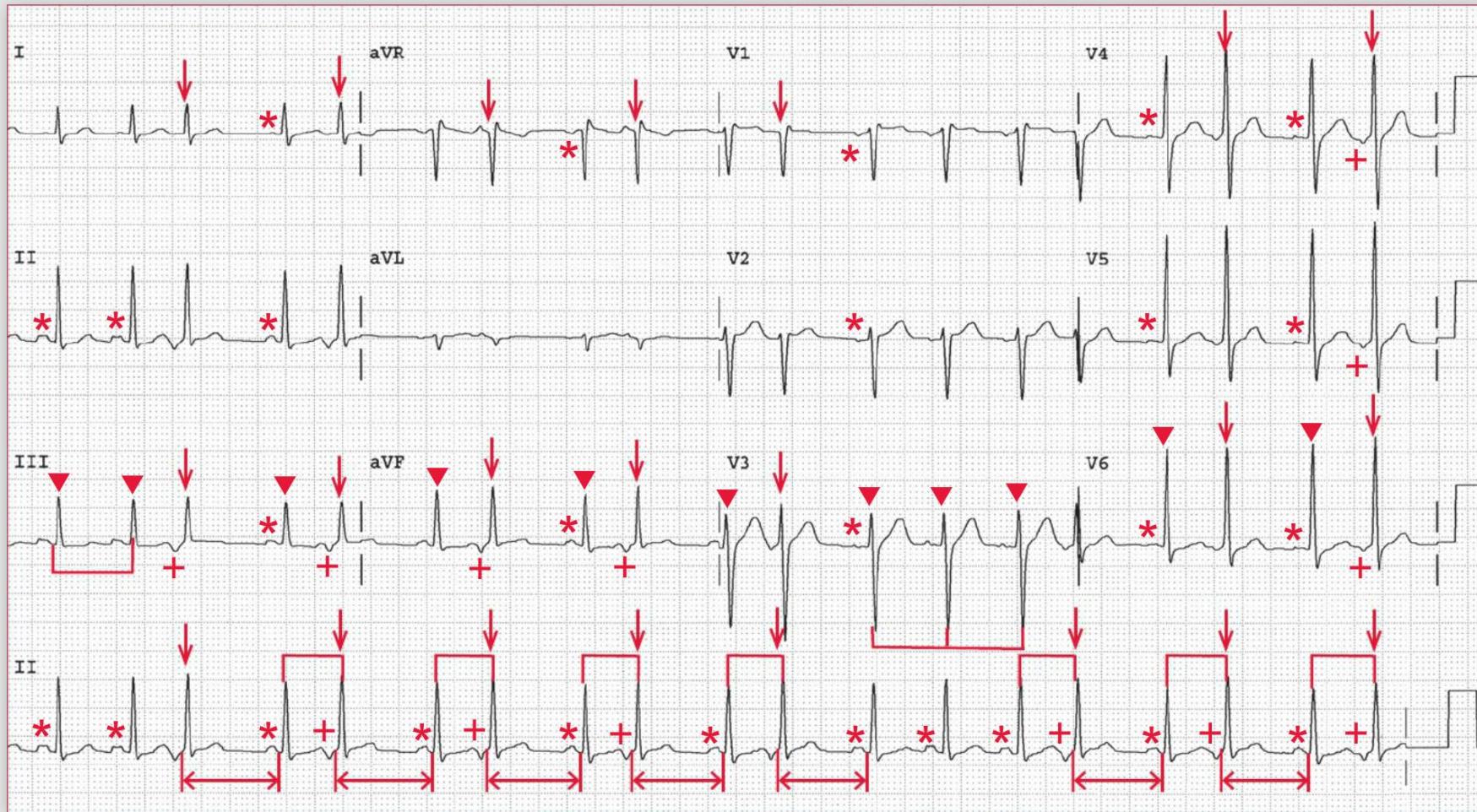
A 35-year-old woman recently diagnosed on echocardiogram with a benign left atrial myxoma causing mitral regurgitation has the following ECG. She does not complain of any symptoms. Plans are under way for resection of the tumor.

What is the rhythm abnormality?

What is the most likely mechanism for this arrhythmia?

How would you manage this patient?





ECG 10 Analysis: Sinus tachycardia with frequent premature atrial complexes (unifocal, full compensatory pause)

The rhythm is irregular, but there is a pattern to the irregularity; that is, all the long intervals (\leftrightarrow) are the same, the short intervals (\square) are the same, and the intermediate intervals (\sqcup) are the same. Hence the rhythm is regularly irregular. There is an underlying regular rhythm (\sqcup) at a rate of 118 bpm (*ie*, the intermediate intervals). There is a P wave (*) before each of the regular QRS complexes (▼) as well as before the QRS complexes that follow the long RR intervals and are associated with the intermediate RR intervals (\sqcup), and the P wave is positive in leads I, II, aVF, and V5-V6; hence this is sinus tachycardia. The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/450 msec).

There are occasional QRS complexes that are early or premature (↓), and before each of these complexes is a P wave (+) that is different than the P waves that originate in the sinus node (*ie*, the P wave is negative in leads II, aVF, and V4-V6). The QRS complex duration, morphology, and axis are identical to those of the sinus QRS complexes. Therefore, these are premature atrial complexes (PACs). Since each PAC has the same abnormal P-wave morphology, these are unifocal PACs.

After the PAC there is a pause (\leftrightarrow) (*ie*, long RR interval), the duration of which is variable. That is, the PP interval surrounding the pause may be shorter than, longer than, or equal to two sinus (PP) intervals. In this case, the PP interval surrounding the pause is equal to two PP intervals. Hence this is called a full compensatory pause. The pause after the PAC is due to the fact that the premature atrial impulse can affect the sinus node, suppressing or resetting its activity. Hence the duration of time before the occurrence of the next sinus impulse is variable. In this case the PAC has suppressed the sinus node, which thereafter generates an on-time P wave.

The most common mechanism of PACs is enhanced automaticity of a specific atrial focus; however, a reentrant circuit within the atria can also cause PACs. PACs occur in individuals of all age groups and can occur in the presence or absence of heart disease. However, the frequency of PACs is higher in patients with structural heart disease, particularly those with left atrial enlargement or hypertrophy, as in the case of mitral valve disease or left ventricular dysfunction. PACs may serve as triggers for other atrial arrhythmias such as atrial fibrillation.

continues

Even though there are no symptoms to suggest a sustained atrial arrhythmia, this patient should undergo Holter monitoring to assess for the presence of other atrial arrhythmias as they may be associated with an increased risk for emboli (particularly atrial fibrillation).

PACs are common, benign, and often asymptomatic, although they can be associated with palpitations or the sensation of skipped beats. No treatment is indicated for the patient who is asymptomatic. However, for symptomatic individuals, the first step in treatment is to avoid precipitating causes such as caffeine, alcohol, smoking, and stress. If

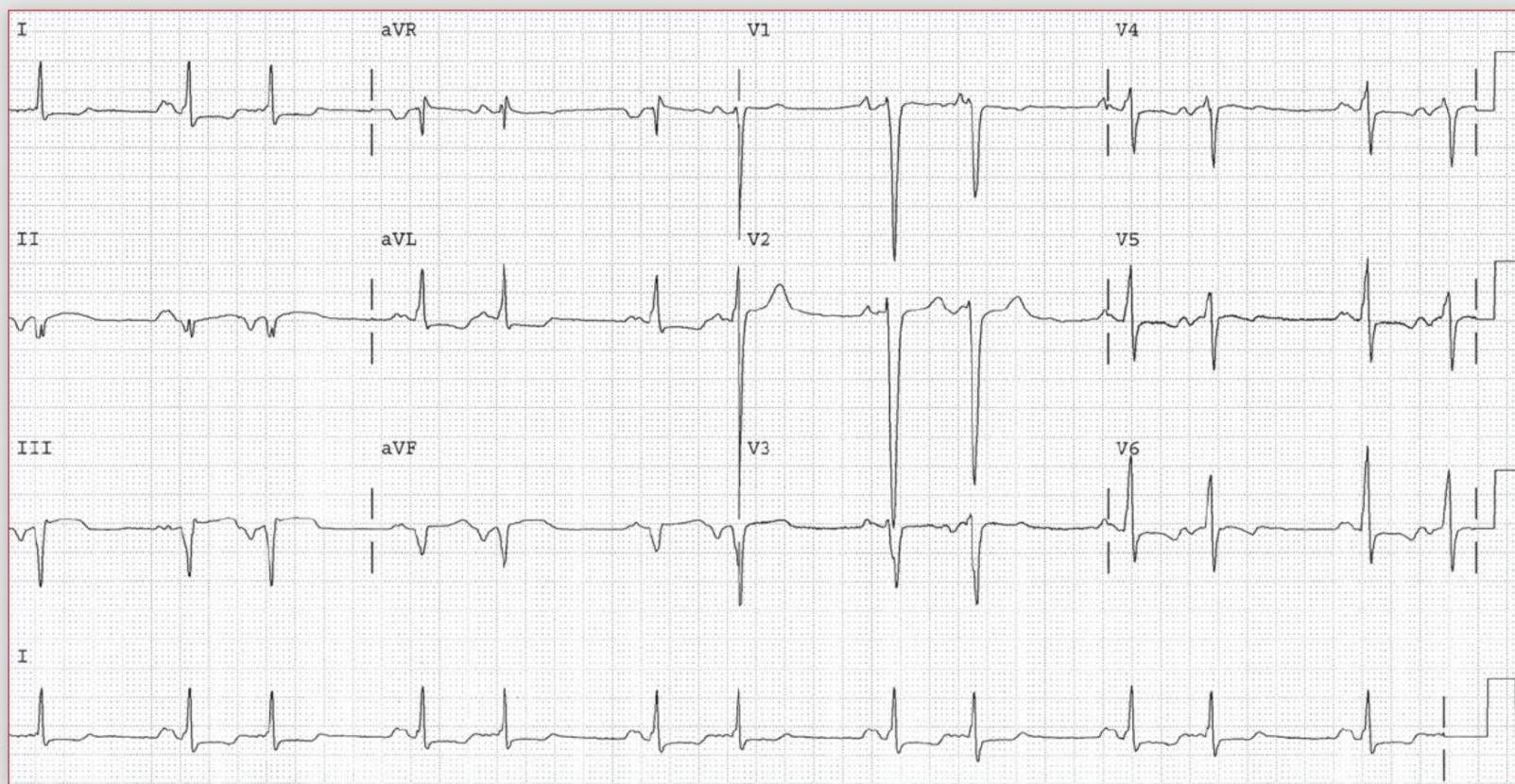
this initial approach is unsuccessful, then a β -blocker can be initiated for treatment of symptoms associated with PACs. A β -blocker does not usually suppress PACs but may reduce symptoms, which are often the result of post-extrasystolic potentiation resulting from the PAC and pause. In this situation there is increased left ventricular filling resulting from the pause, and this will cause an increase in inotropy (*ie*, the Frank-Starling effect). Suppressive therapy with an anti-arrhythmic drug (class IA, IC, or III) may be necessary if the PACs are associated with symptoms that persist despite therapy with a β -blocker or if they are “triggers” for sustained atrial arrhythmias. ■

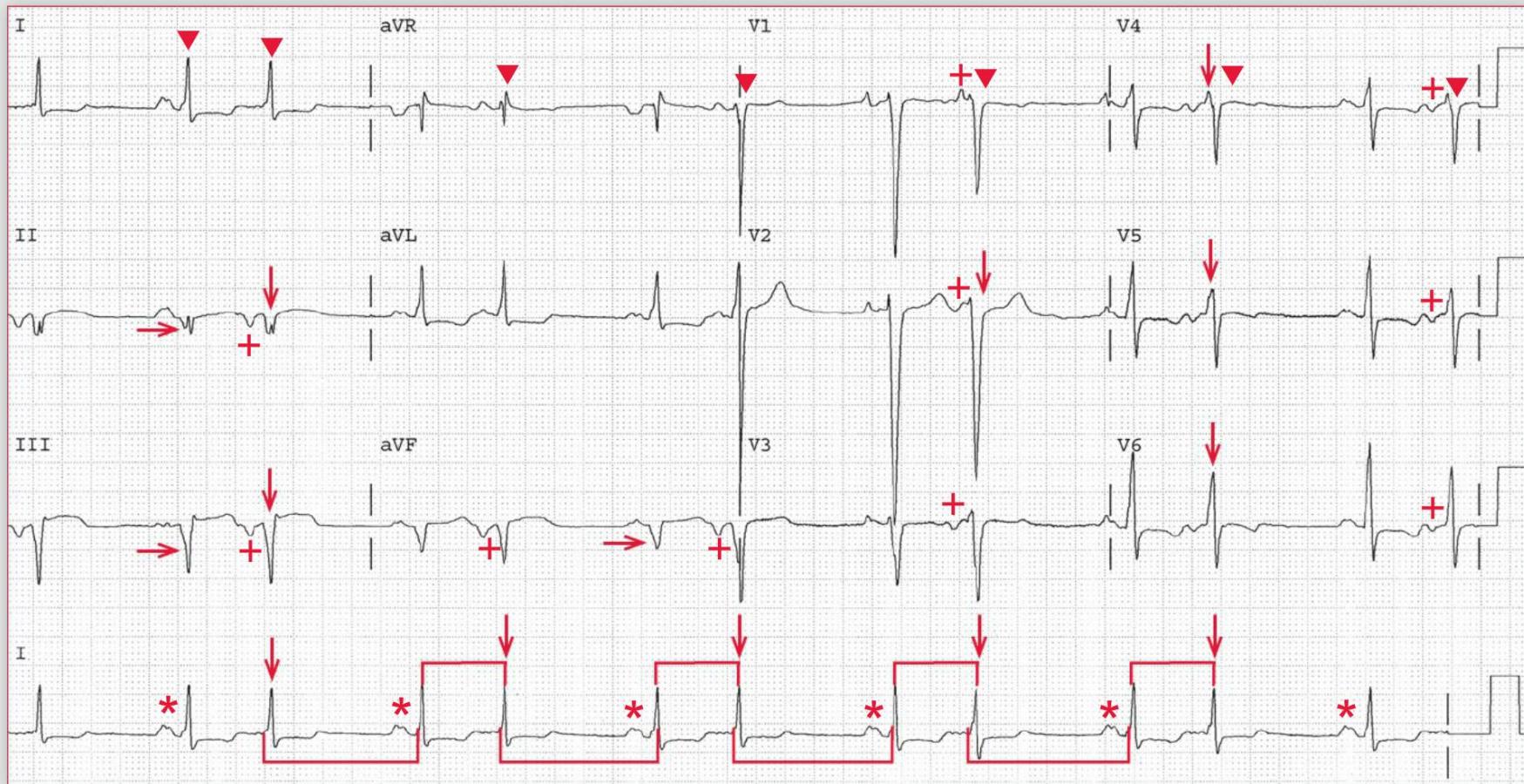
A 52-year-old woman with known coronary artery disease and normal left ventricular function presents for a routine office visit. She reports the recent onset of intermittent palpitations. On examination, her pulse is irregular and her blood pressure is 125/80 mm Hg. Jugular venous pressure is normal. Cardiac exam reveals a mid-systolic click with a late systolic murmur best heard at the apex. The rest of her exam is unremarkable. You obtain the following ECG.

What is the rhythm abnormality?

Is any therapy indicated?

What is the clinical diagnosis?





ECG 11 Analysis: Sinus rhythm with atrial bigeminy, left atrial hypertrophy, intraventricular conduction delay, prior inferior wall myocardial infarction, clockwise rotation (poor R-wave progression, late transition)

The rhythm is irregular at an average rate of 66 bpm. However, there is a pattern of long (◻) and short (◻) intervals (group beating); hence the rhythm is regularly irregular. After the long interval there is a P wave (*), which is the same before each of the QRS complexes (▼) that follow each long RR interval, and it has a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V5; hence this is a sinus complex. The P wave is broad in leads I, II, aVF, and V5-V6 (0.16 sec), and slight notching can be seen. This is consistent with left atrial hypertrophy or left atrial abnormality. The shorter RR interval is the result of a premature complex (↓) that is preceded by a P wave (+), but the P-wave morphology is different than that of the sinus P wave; it is negative in leads II, aVF, and V4-V6. Hence these are premature atrial complexes (PACs) (↓). There is a repeating pattern, and every other QRS complex is a PAC. This is termed atrial bigeminy or PACs in a bigeminal pattern, indicating that there is a repeating pattern of the premature complexes, and has no clinical implications.

The QRS complex duration is prolonged (0.11 sec) as a result of an intraventricular conduction delay. The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, the negative QRS complex is the result of deep Q waves in leads II, III, and aVF (→). Hence this is an inferior wall myocardial infarction and not a conduction abnormality (*ie*, left anterior fascicular block in which the QRS complex in leads II and aVF has an rS morphology). Also noted is poor R-wave progression from leads V1 to V4, with QRS complex transition occurring in lead V5. This is termed clockwise rotation and is due to a change in the electrical axis in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. With clockwise rotation, left ventricular forces develop late in the left precordial leads. The QT/QTc intervals are normal (460/480 sec and 430/450 msec when the prolonged QRS complex duration is considered).

continues

The presence of atrial bigeminy does not have any important clinical implications and is indicative of the presence of frequent PACs. No specific therapy is necessary for this arrhythmia. Symptoms, including palpitations or skipped beats, however, may occur. Palpitations are generally the result of increased inotropy and stroke volume resulting from the pause and increased left ventricular filling (Frank-Starling mechanism). β -blockers may be effective in alleviating the symptoms (via their negative inotropic effect), although they generally do not result in suppression of the premature complexes.

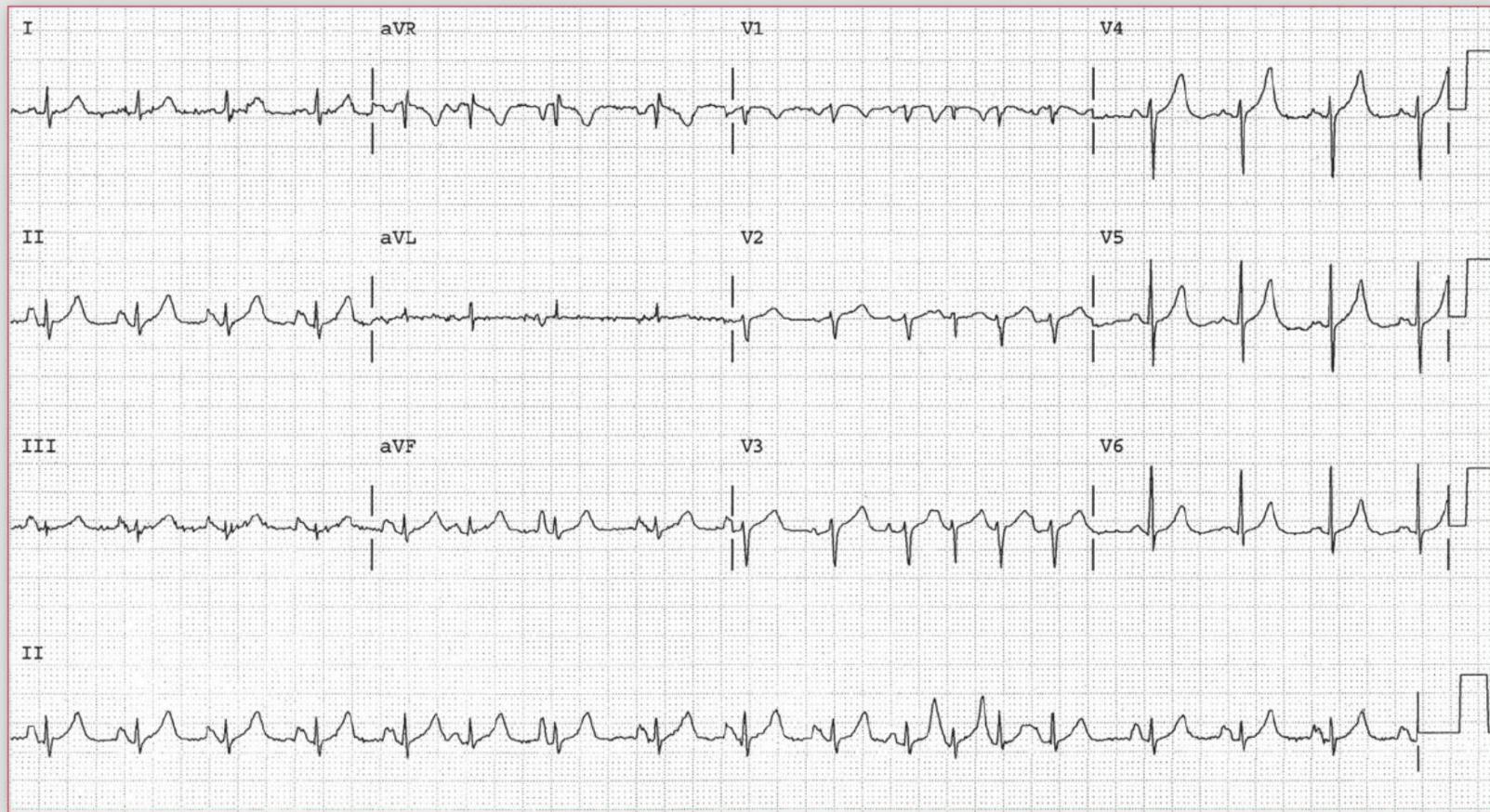
Mitral valve prolapse (MVP) involves myxomatous thickening of the mitral valve leaflets resulting in redundant tissue that slips or billows back into the left atrium during systole. This can lead to a spectrum of

mitral regurgitation ranging from insignificant to severe. MVP is present in roughly 2% to 3% of the population and can be found either in isolation or in association with other connective tissue disorders such as Marfan syndrome. Diagnosis can be made by physical examination or echocardiography. On auscultation, the prolapse of the mitral valve results in a mid-systolic click followed by a late systolic murmur from the mitral regurgitation. MVP has been associated with infective endocarditis, ischemic stroke, and both atrial and ventricular arrhythmias, including PACs and atrial fibrillation. Treatment of the arrhythmias in symptomatic individuals consists of avoidance of potential precipitants (such as caffeine and alcohol) and/or initiation of nodal agents such as a β -blocker. Corrective surgery can be performed in the case of severe mitral regurgitation. ■

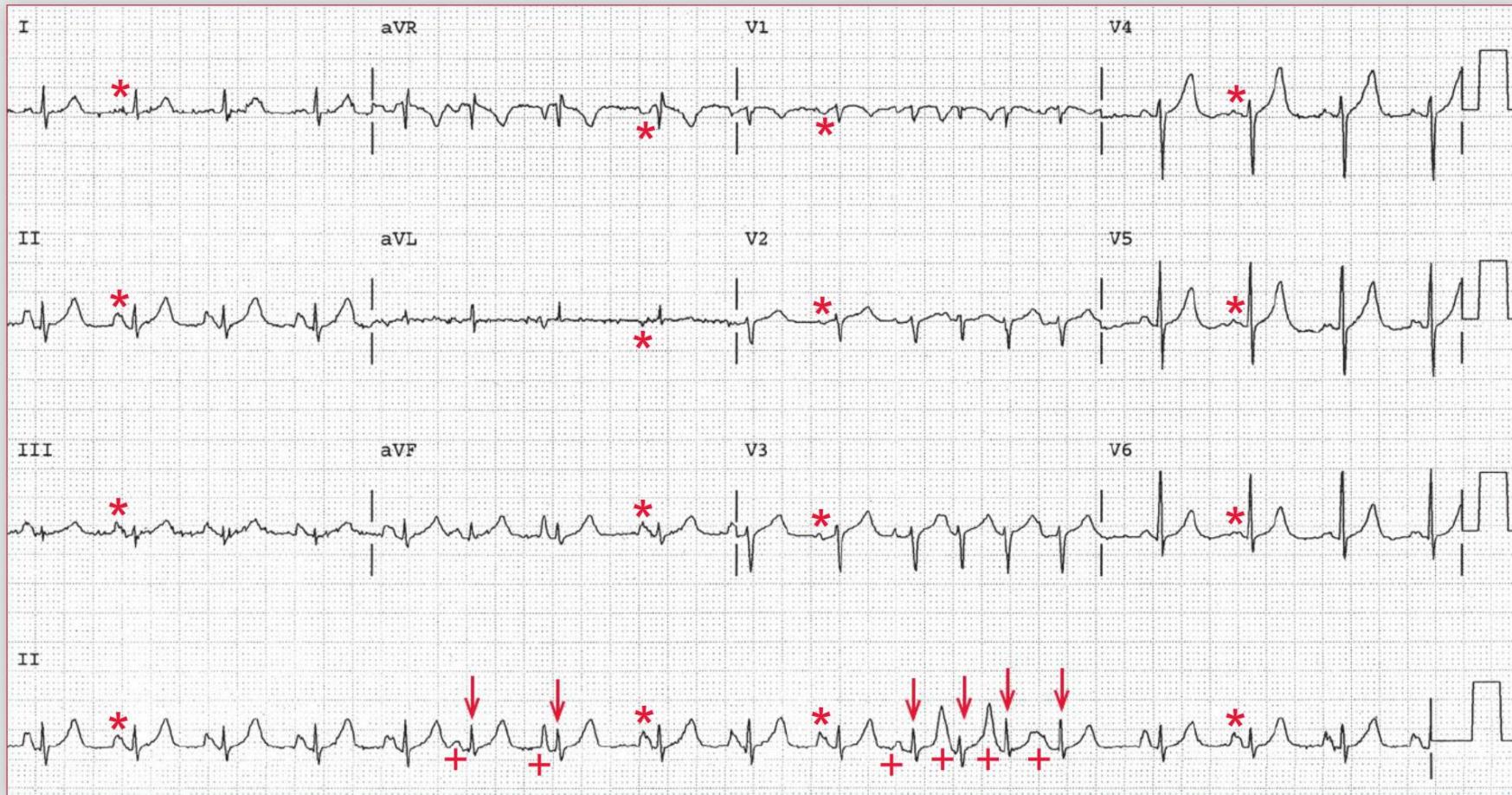
A 65-year-old man with a 40 pack-year smoking history presents to his primary care physician for a routine health maintenance visit. His doctor notes that his heart sounds are irregular and distant. The physician obtains the ECG shown below.

What are the abnormalities?

What is likely to be the underlying disease?



Podrid's Real-World ECGs



ECG 12 Analysis: Normal sinus rhythm, multifocal premature atrial complexes, low-voltage limb leads, clockwise rotation (poor R-wave progression, late transition)

The rhythm is initially regular at a rate of 96 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. However, there are occasional QRS complexes that are early (premature) (*ie*, the sixth, seventh, and 11th through 14th complexes) (↓). There is a P wave (+) in front of these premature complexes; however, there are various P-wave morphologies, all of which are different than the morphology of the sinus P wave. These are, therefore, termed multifocal premature atrial complexes.

In addition, there is low voltage in the limb leads (R wave < 5 mm in amplitude in each lead) and poor R-wave progression across the precordium with late transition (R/S > 1 in lead V5). This is the result of clockwise rotation of the electrical axis in the horizontal plane, which

is determined by imagining the heart as viewed from under the diaphragm. With clockwise rotation the left ventricular forces are seen later, in the lateral precordial leads.

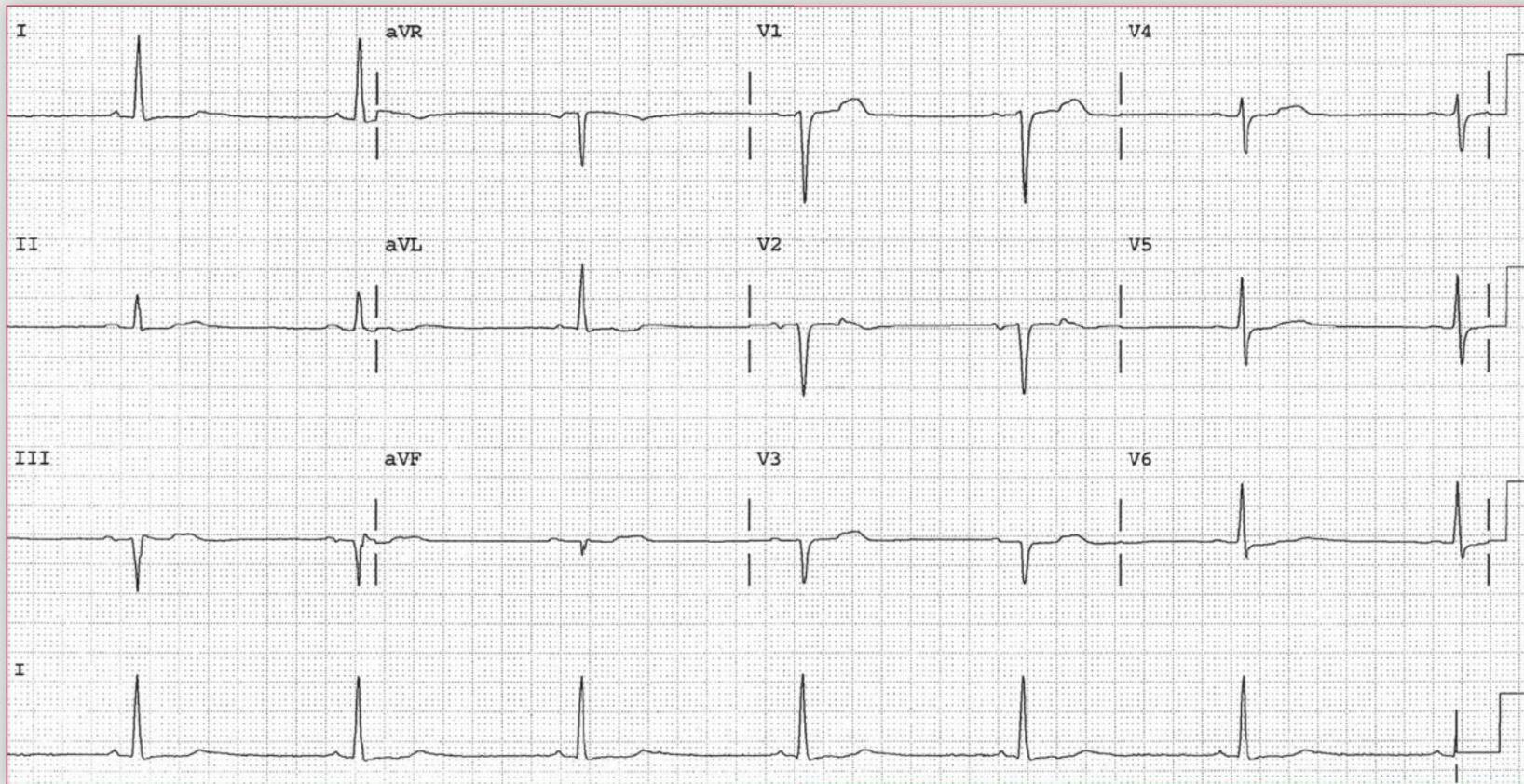
The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/405 msec).

This patient likely has chronic obstructive pulmonary disease that has resulted in hyperinflation of the chest and resultant clockwise rotation of the heart. This explains the poor R-wave progression and low voltage in the limb leads. In addition, atrial ectopy is common in chronic pulmonary disease. ■

Core Case 13

A 53-year-old woman with mitral valve prolapse is noted to have bradycardia at her first visit with a new internist. She has no current symptoms.

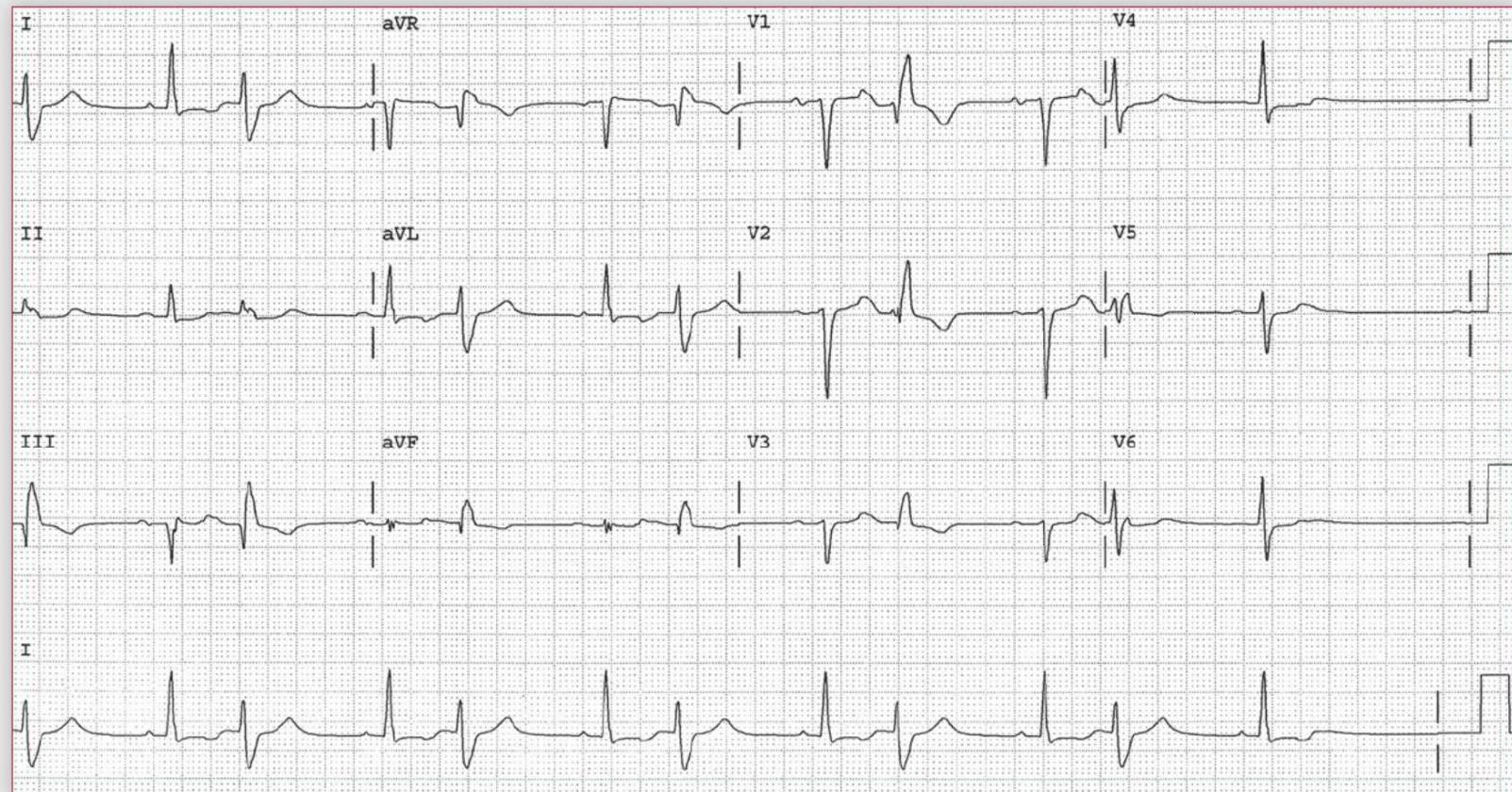
ECG 13A

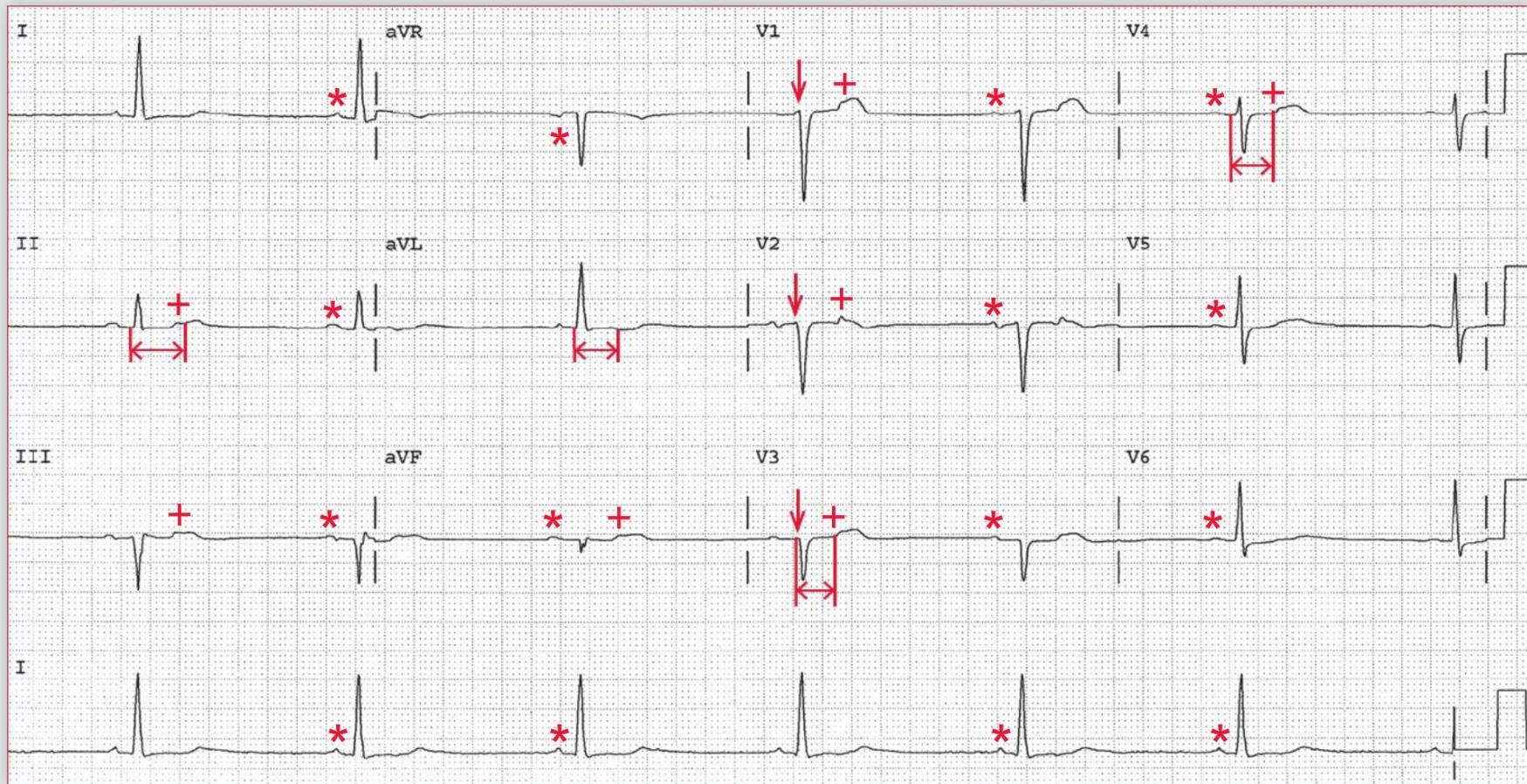


An ECG is obtained (ECG 13A). As a result of the bradycardia, she is referred to a cardiologist, who obtains another ECG (13B).

What do her ECGs show?

ECG 13B





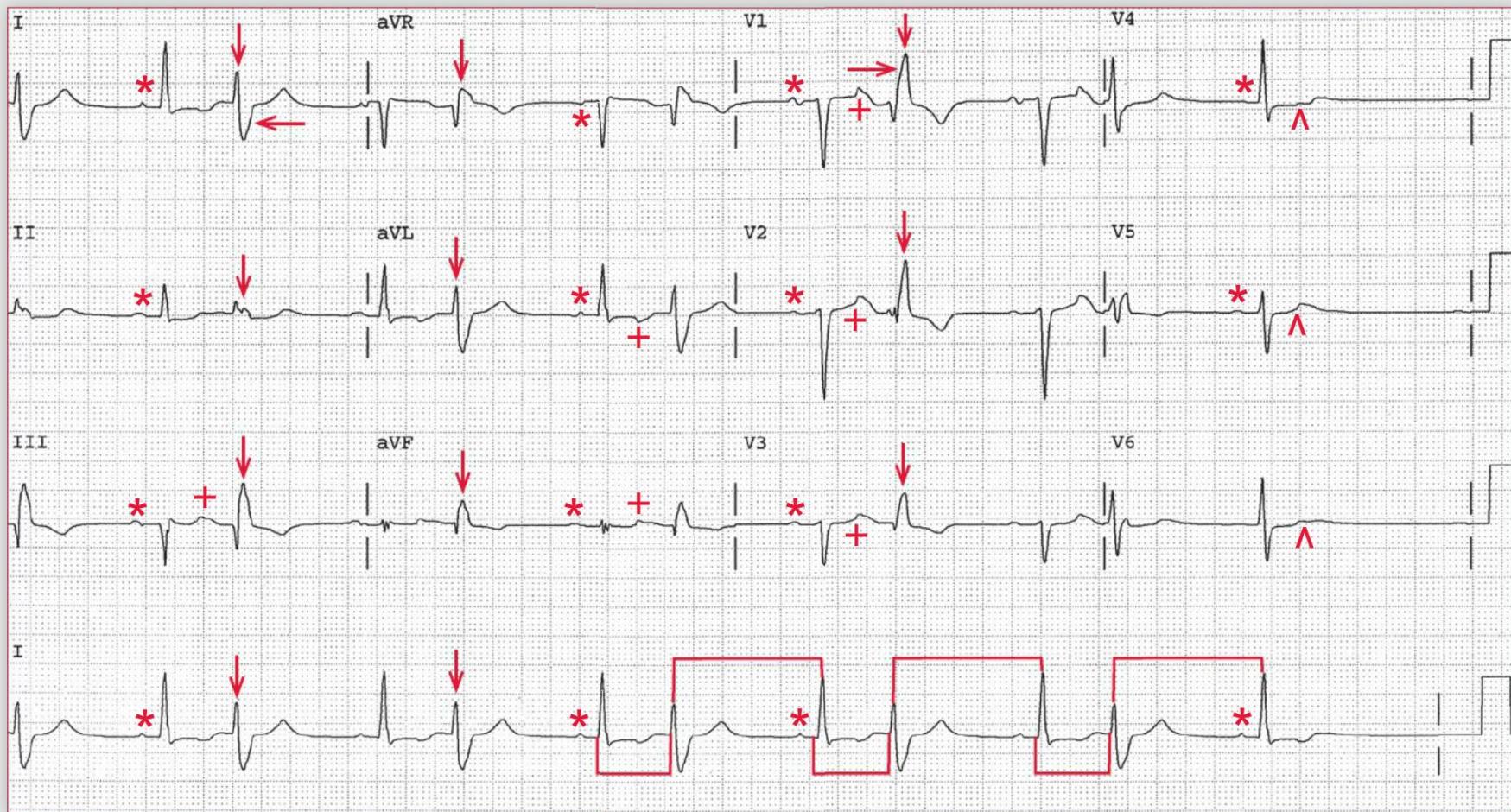
ECG 13A Analysis: Sinus rhythm, atrial bigeminy, blocked premature atrial complexes (blocked premature atrial complexes in a bigeminal pattern), left axis, old anteroseptal wall myocardial infarction

The rhythm in ECG 13A is regular at a rate of 40 bpm. There is a P wave (*) in front of each QRS complex with a constant PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence there is a sinus rhythm present. Within each T wave is a notching that is best appreciated in leads II, III, aVF, and V1-V4 (+). The normal T wave has a smooth upstroke and downstroke. Any bumps or notches on the T waves are probably superimposed P waves. Hence these are early (premature) P waves that have a morphology that is different than the sinus P waves and do not result in a ventricular complex (*ie*, they are nonconducted). These are termed blocked premature atrial complexes (PACs) and, as every other P wave is a blocked PAC, this is a bigeminal

pattern. There is a fixed relationship or coupling interval between the sinus P wave and premature P wave (\leftrightarrow). As a result of the blocked PACs, the effective heart rate is slow (*ie*, 40 bpm).

The QRS complex duration (0.08 sec) is normal. The axis is leftward, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF); this is a physiologic left axis. The QT/QTc intervals are normal (440/360 msec). There are no R waves in leads V1-V3 (\downarrow), consistent with an old anteroseptal wall myocardial infarction.

continues



ECG 13B Analysis: Sinus rhythm, atrial bigeminy,
rate-related right bundle branch block

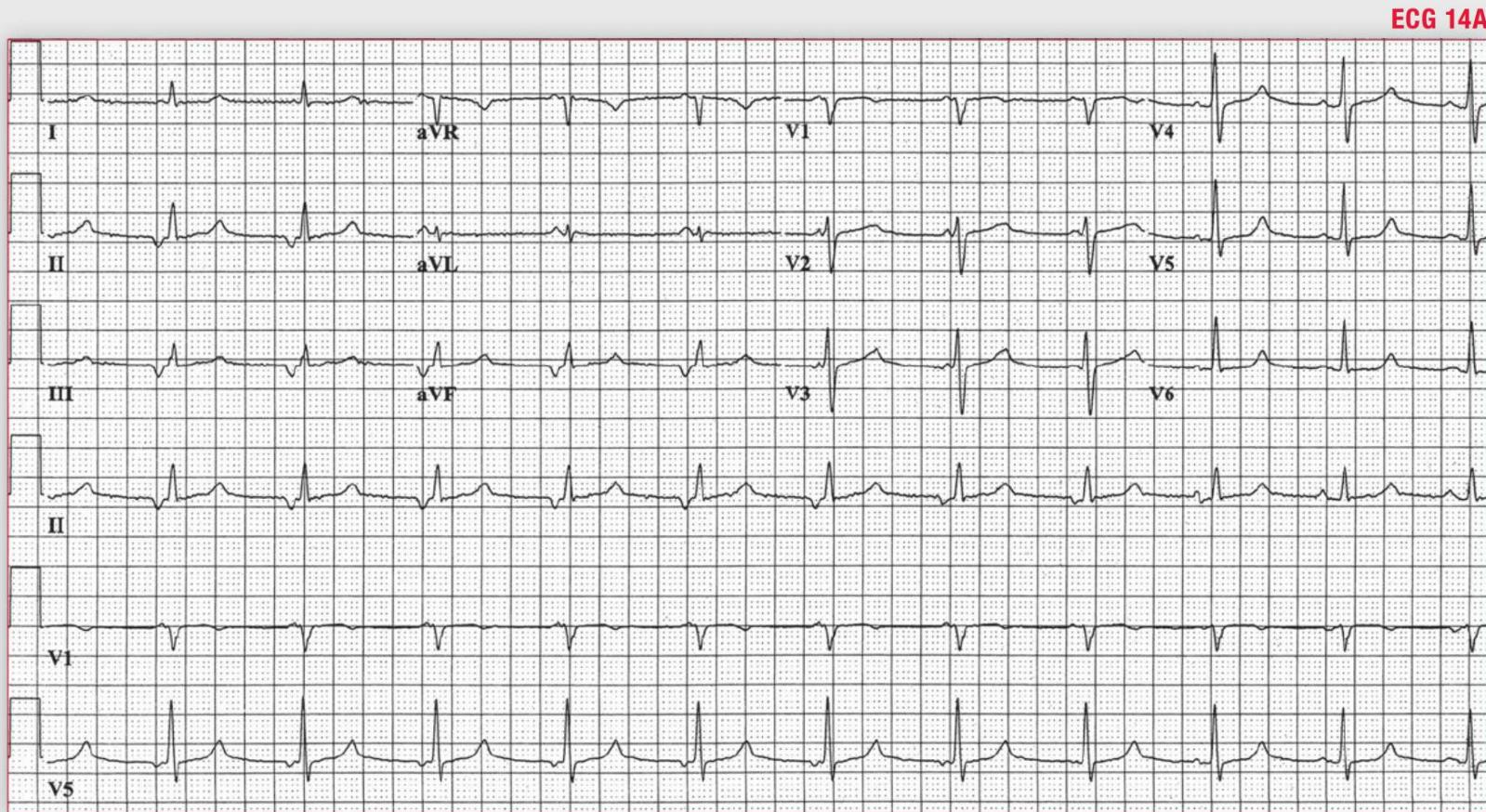
ECG 13B shows a regularly irregular rhythm at a rate of 48 bpm. There is a pattern of group beating, with alternating long (□) and short (□) RR intervals. A P wave (*) can be seen before each of the narrow QRS complexes (which have the same duration, morphology, and axis as the QRS complexes in ECG 13A) with a constant PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. Every other QRS complex is early (↓) and wide (0.16 sec) with a right bundle branch block morphology (RSR' morphology in lead V1 [→] and broad S wave in lead I [←]). Prior to each of these wide QRS complexes there is also a P wave (+), which is early and can be seen as a notching within the T wave (especially in leads V1-V3) (+), similar to the notching of the T wave seen in ECG 13A. The relationship between the sinus P wave and premature

P wave is constant; that is, there is a fixed coupling relationship. These are PACs occurring in a bigeminal pattern. Unlike the blocked complexes in ECG 13A, the premature P wave results in a QRS complex (*ie*, there is AV conduction). The early QRS complex is with a right bundle branch block, which is a rate-related aberrancy. After the last QRS complex, the premature P wave (Λ) is again blocked as there is no QRS complex that follows.

Blocked or nonconducted PACs do not require therapy unless they are associated with symptomatic bradycardia. In this case, they may be suppressed with a class IA, IC, or III anti-arrhythmic agent. Likewise, the conducted PACs are common, benign, and do not require therapy unless associated with significant symptoms. ■

Core Case 14

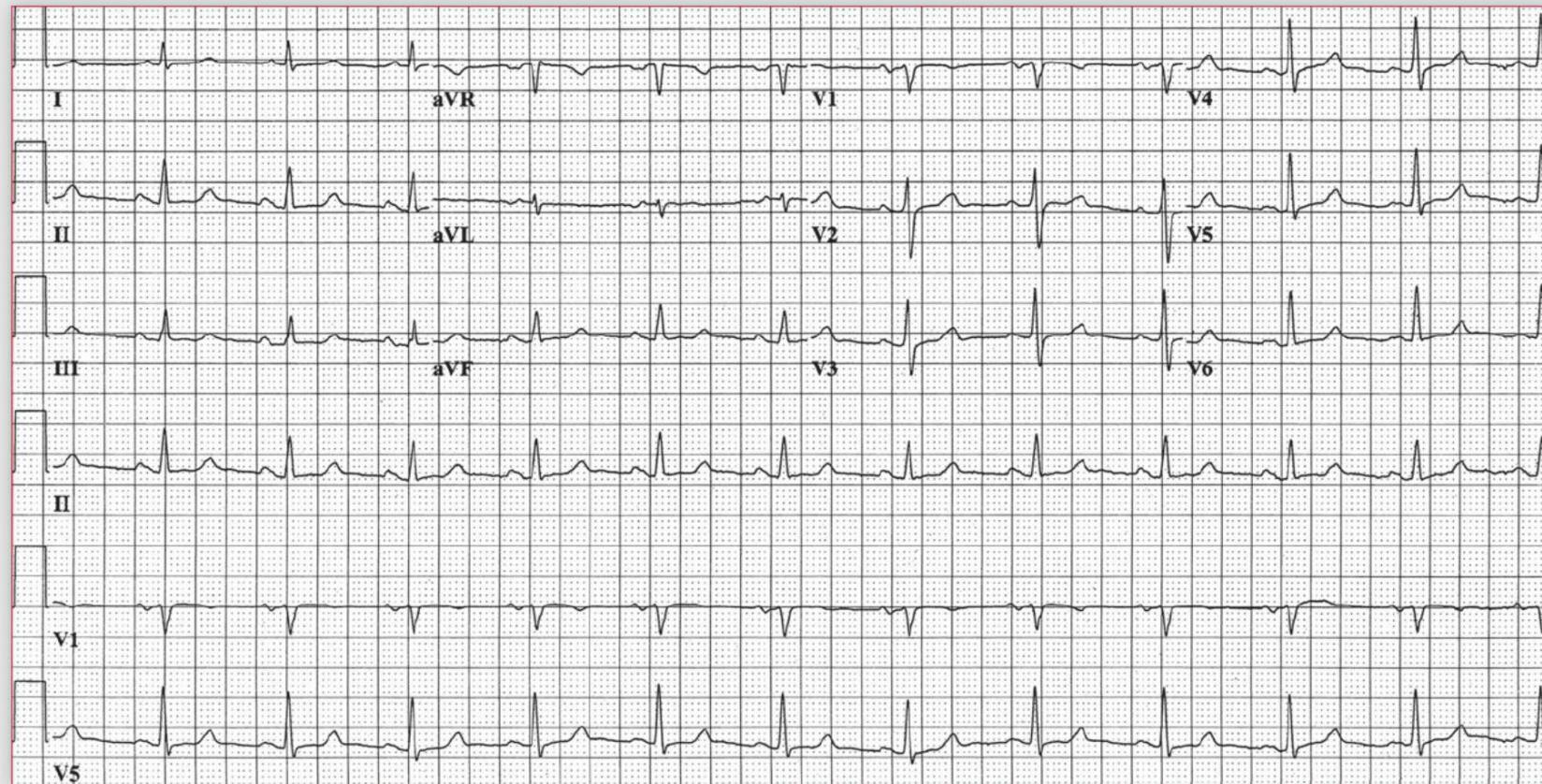
A 34-year-old man presents to the emergency department with chest pain after inhaling cocaine. His pain has fully resolved by the time he reaches the

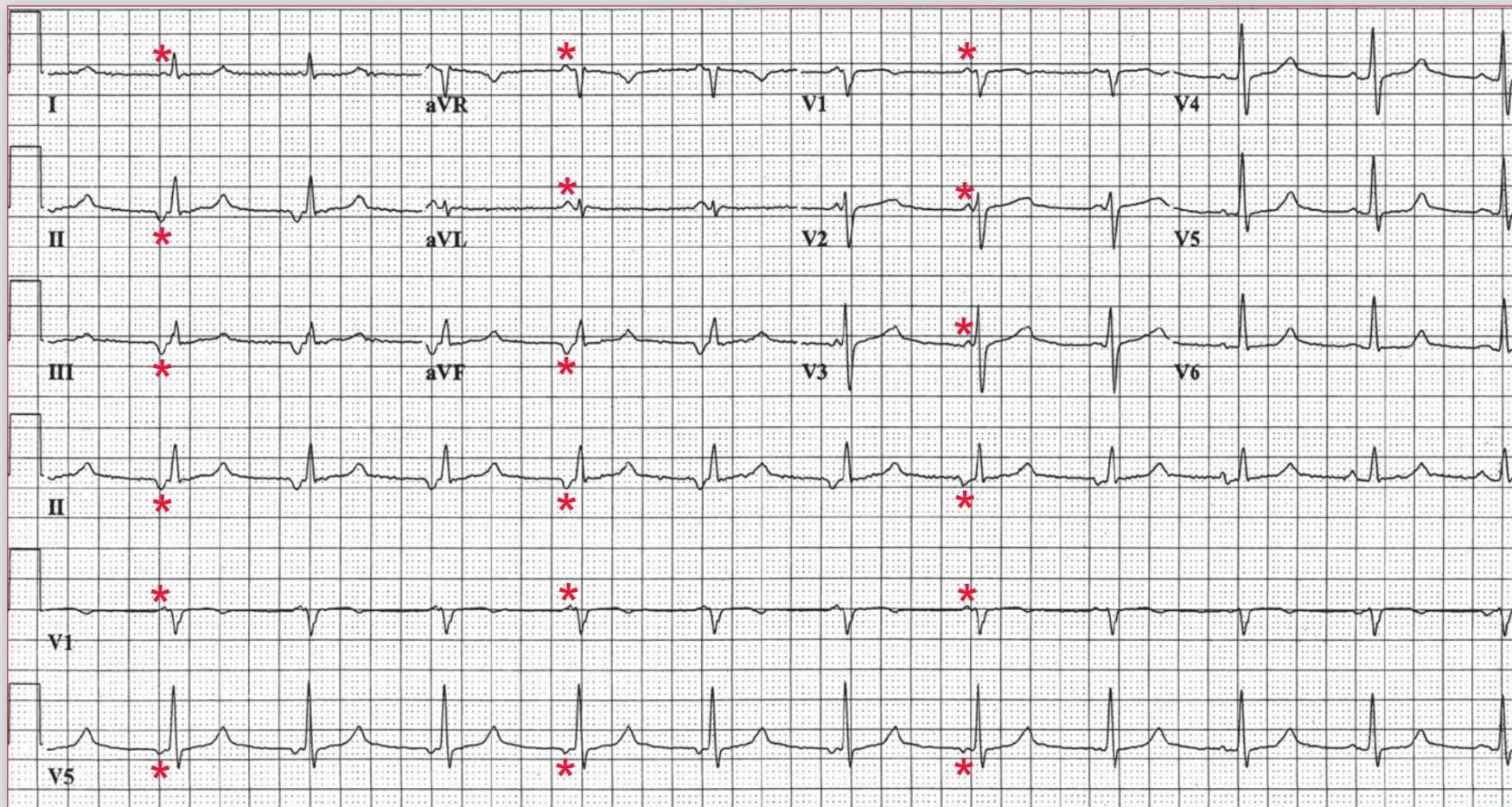


emergency department, and he is generally feeling better. His presenting ECG is shown (14A). A second ECG is obtained 2 hours later (ECG 14B).

What is the abnormality in ECG 14A?
What is the likely cause of this abnormality?
What does ECG 14B show?

ECG 14B





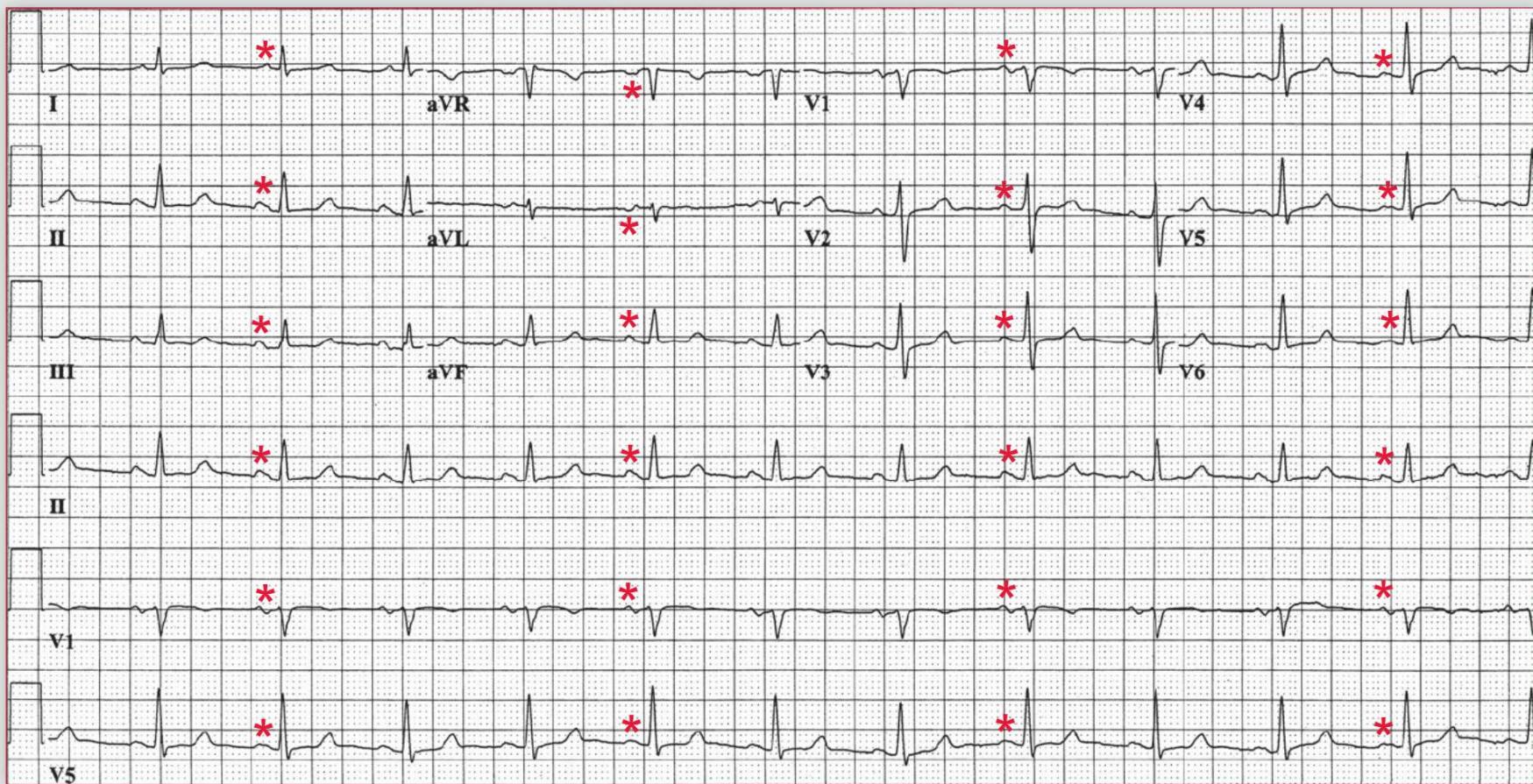
ECG 14A Analysis: Ectopic atrial rhythm

In ECG 14A there is a regular rhythm at a rate of 66 bpm. A P wave (*) is present before each QRS complex with a constant PR interval (0.12 sec). However, the P wave is negative (inverted) in leads II and aVF. It is, therefore, not a sinus P wave and the rhythm is not sinus. It is an ectopic atrial focus and hence this is an atrial rhythm associated with a short PR interval. The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/420 msec).

An ectopic atrial rhythm is identified by the presence of distinct P waves of uniform morphology before each QRS complex. However, the P wave differs from that of sinus rhythm (it is inverted or biphasic). The PR interval is constant and may be the same as or different than that of sinus rhythm. The QRS intervals are regular.

Ectopic atrial rhythms arise when an ectopic focus within the atrial myocardium becomes active and generates an action potential at a rate faster than the sinus node. This can arise from elevated sympathetic nervous system output or from drugs that increase sympathetic tone (eg, caffeine, cocaine). In this case, the ectopic atrial rhythm may have been triggered by cocaine use. Alternatively, there may be depression of sinus node automaticity, allowing an ectopic atrial focus to become manifest.

continues

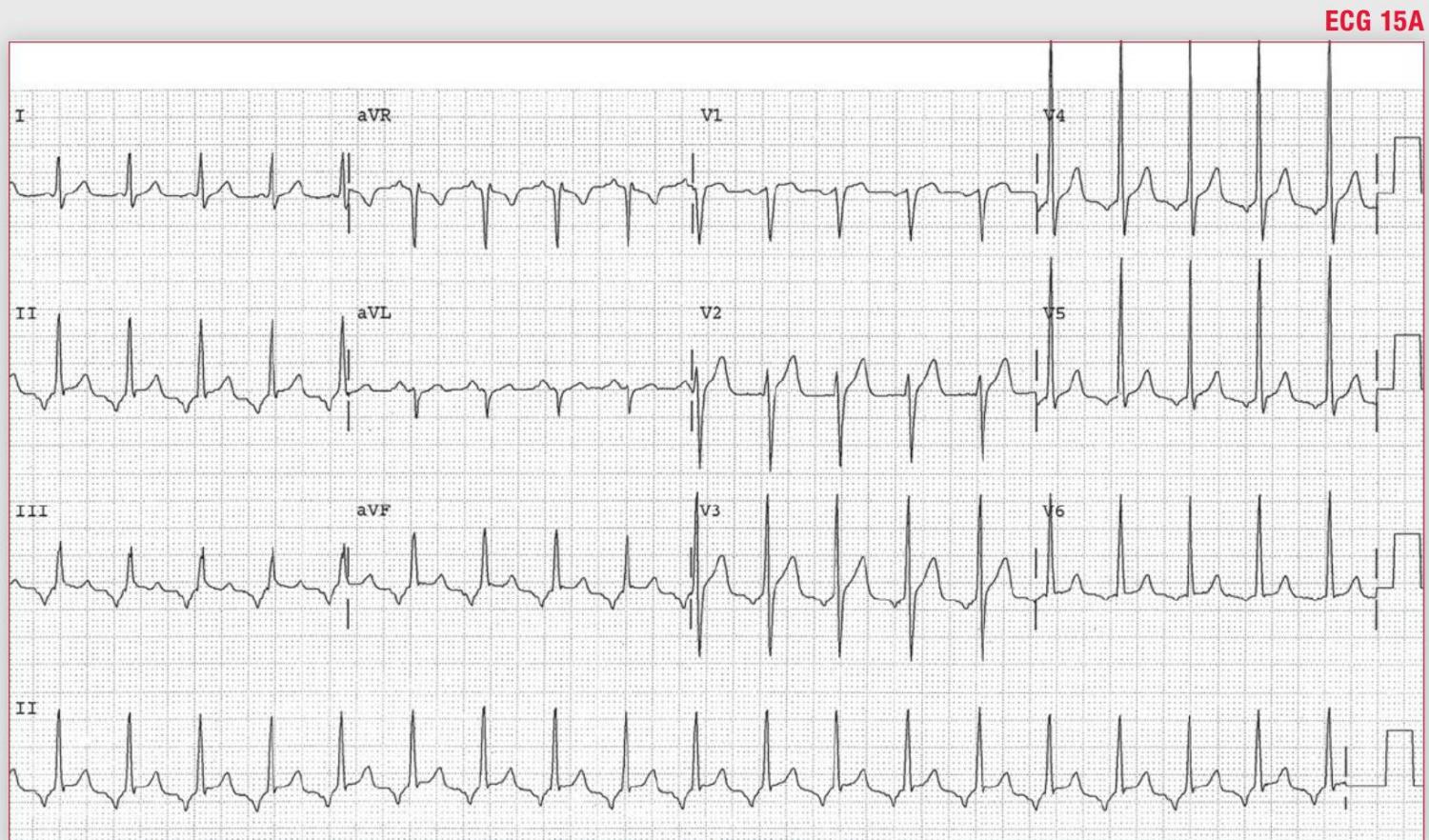


ECG 14B Analysis: Normal sinus rhythm

ECG 14B shows a regular rhythm at a rate of 72 bpm. Noted are P waves (*) before each QRS complex with a stable PR interval (0.16 sec). The QRS complex duration, axis, and morphology and QT/QTc intervals are the same as in ECG 14A. The P waves are positive in leads I, II, aVF, and V4-V6. This is, therefore, a normal sinus rhythm. Of note, the rate is slightly faster than the rate in ECG 14A. Therefore, the ectopic atrial focus may still be capable of spontaneous activity, but it is now suppressed by a faster sinus rate. ■

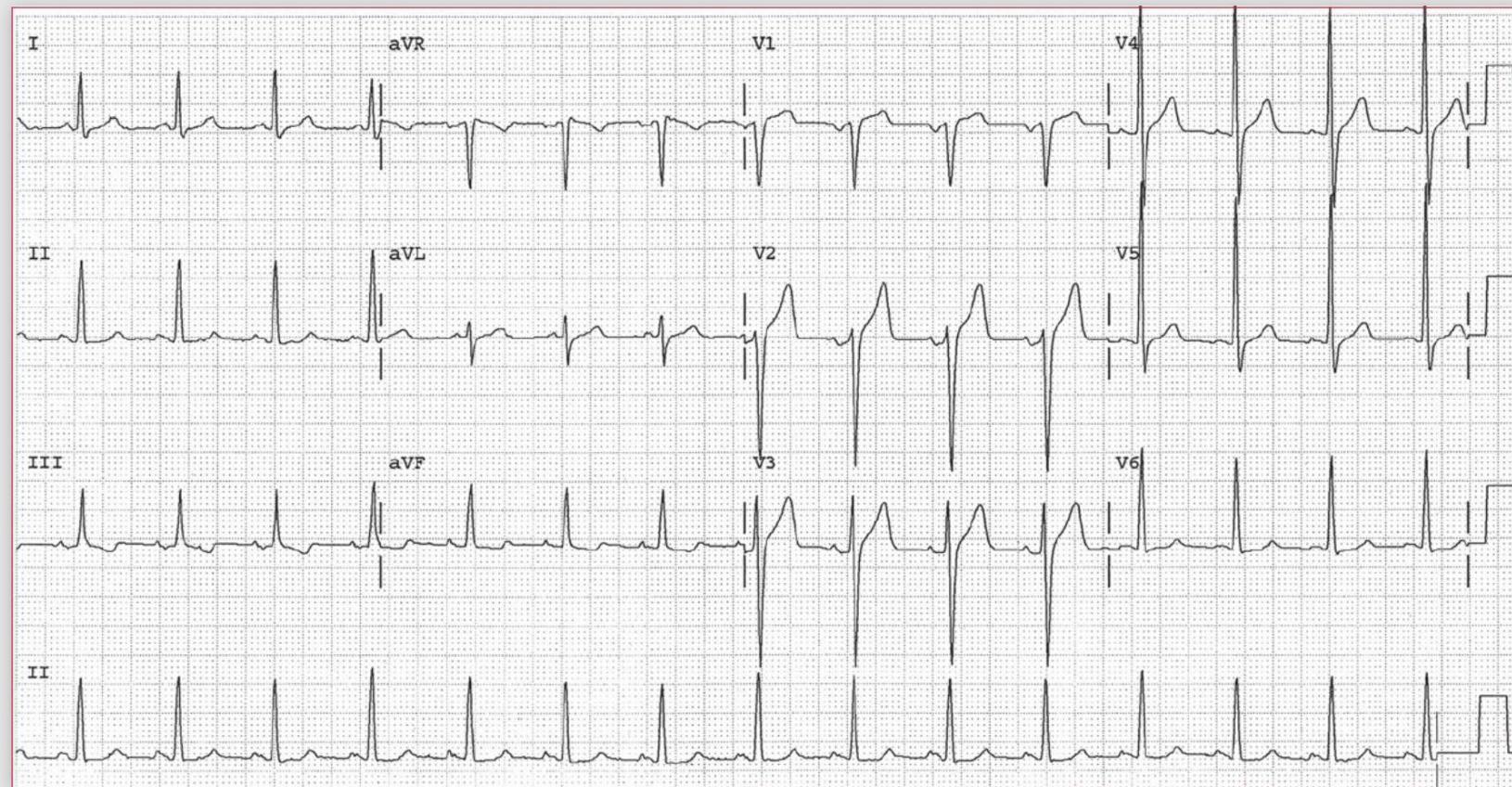
Core Case 15

A 34-year-old woman with non-ischemic cardiomyopathy presents to the emergency department with palpitations. The palpitations have been present for about 24 hours and have been incessant. She has not had any syncope or pre-syncope,



but she is feeling progressively fatigued. The emergency department physician administers an intravenous β -blocker, and an ECG is obtained (ECG 15A). About 10 minutes later, the patient's symptoms abate and a second ECG is obtained (ECG 15B).

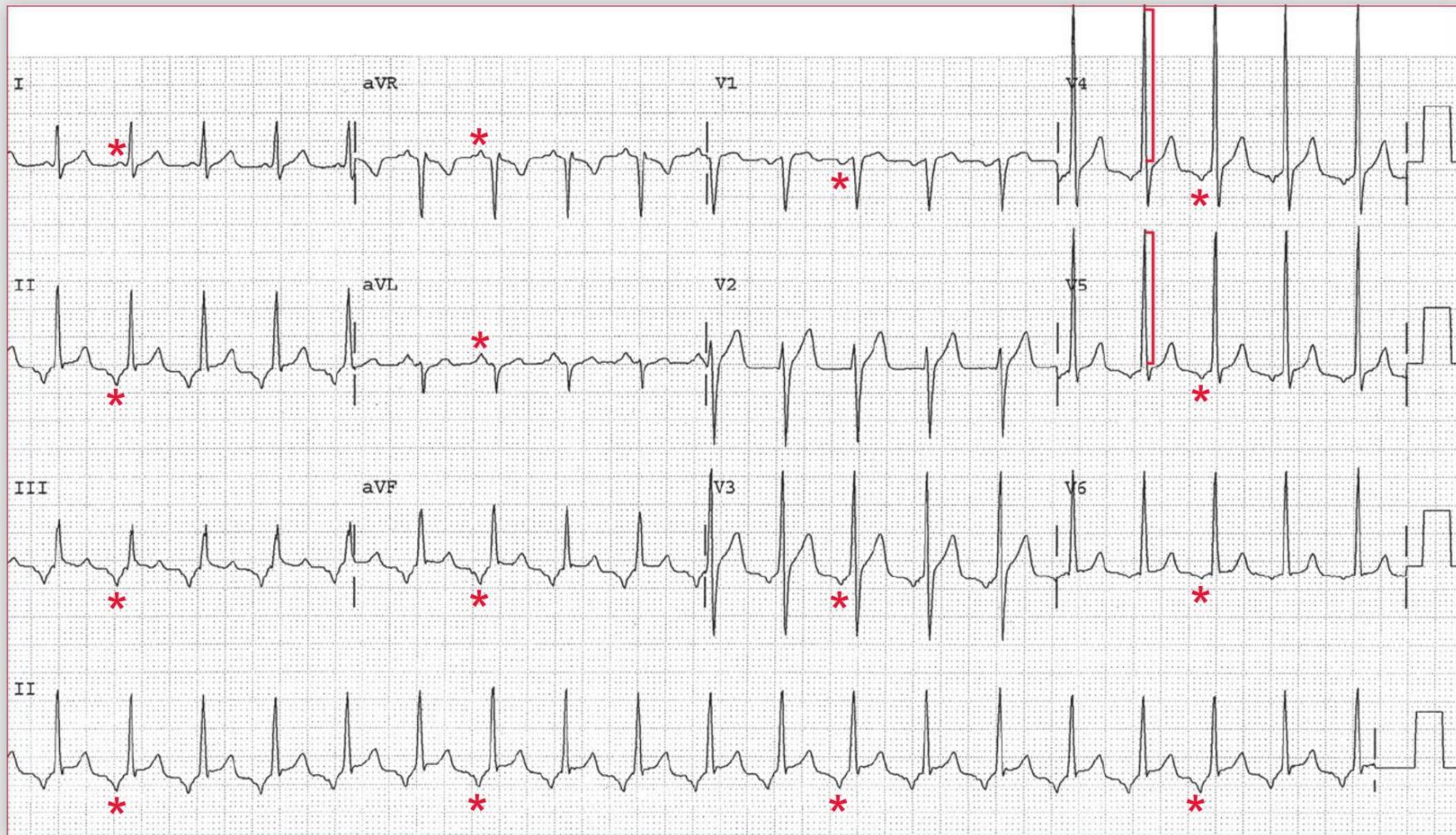
ECG 15B



What does her presenting ECG (15A) show?

What is the etiology?

What does the second ECG (15B) show?



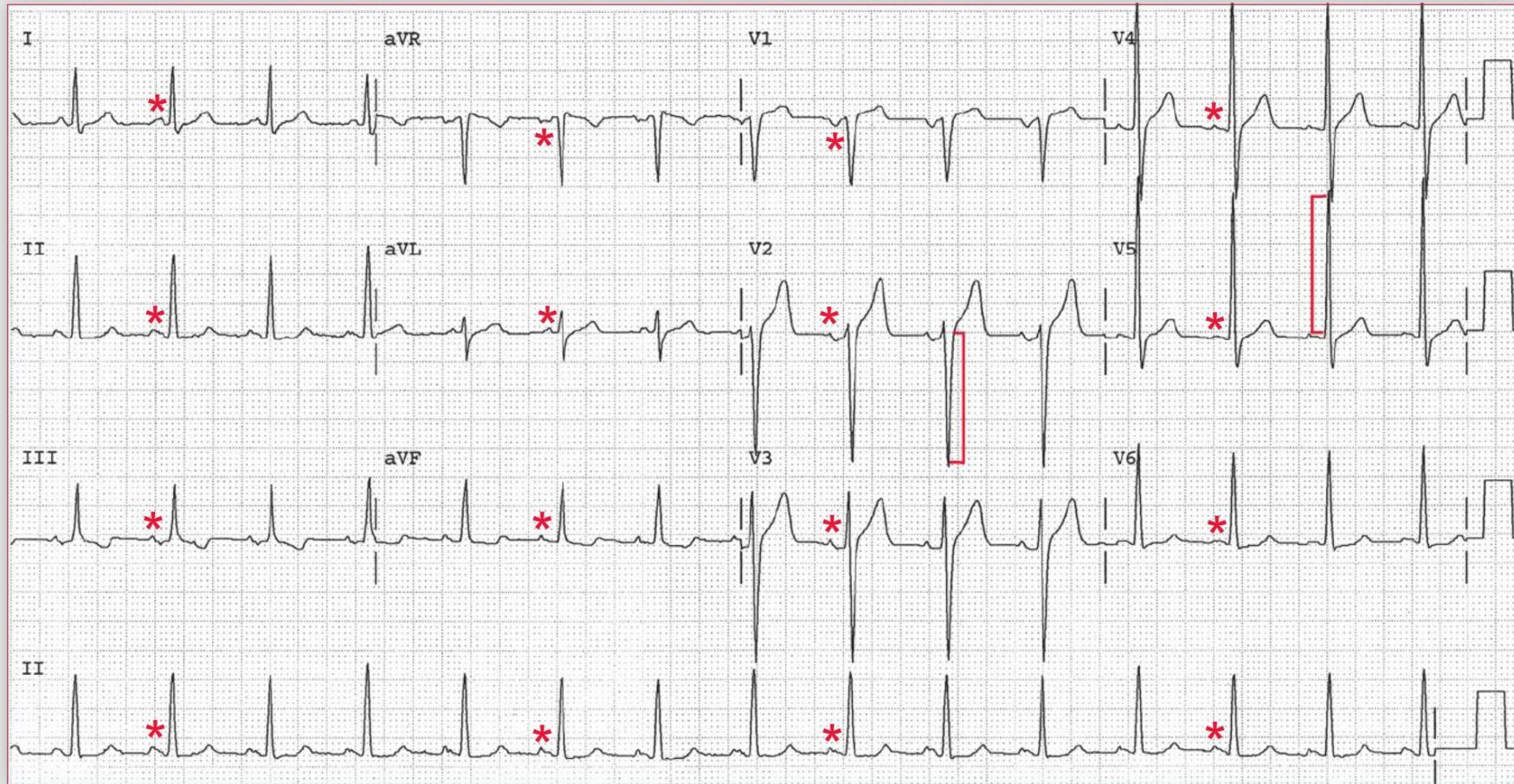
ECG 15A Analysis: Ectopic atrial tachycardia, left ventricular hypertrophy (LVH)

In ECG 15A there is a regular rhythm at a rate of 126 bpm. A P wave (*) is present before each QRS complex with a stable PR interval (0.14 sec). The P wave is negative (inverted) in leads II, aVF, and V3-V6. Therefore, the atrial impulse is being initiated from an ectopic atrial focus and not the sinus node. This is atrial tachycardia.

The QRS complex duration (0.08 sec) and morphology are normal. The QT/QTc intervals are normal (280/400 msec). The QRS amplitude () in leads V4-V5 is high (28 mm), consistent with left ventricular hypertrophy. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF).

continues

Podrid's Real-World ECGs



ECG 15B Analysis: Normal sinus rhythm, LVH

In ECG 15B the QRS complex duration, morphology, and axis are the same as in ECG 15A. There is a regular rhythm at a rate of 90 bpm. The QT/QTc intervals are normal (320/390 msec). There is a P wave (*) before each QRS complex with a fixed PR interval (0.16 sec). The P wave is positive (upright) in leads I, II, aVF, and V4-V6, and hence the impulse is originating from the sinus node. This is, therefore, a normal sinus rhythm. The QRS complex duration and morphology are normal. Together, the S-wave depth in lead V2 () and the R-wave amplitude in lead V5 () total 45 mm, which is consistent with left ventricular hypertrophy.

Most commonly, atrial tachycardia is the result of rapid impulse generation from an ectopic atrial focus. It has the same mechanism as an ectopic atrial rhythm but occurs at a faster rate (*ie*, > 100 bpm).

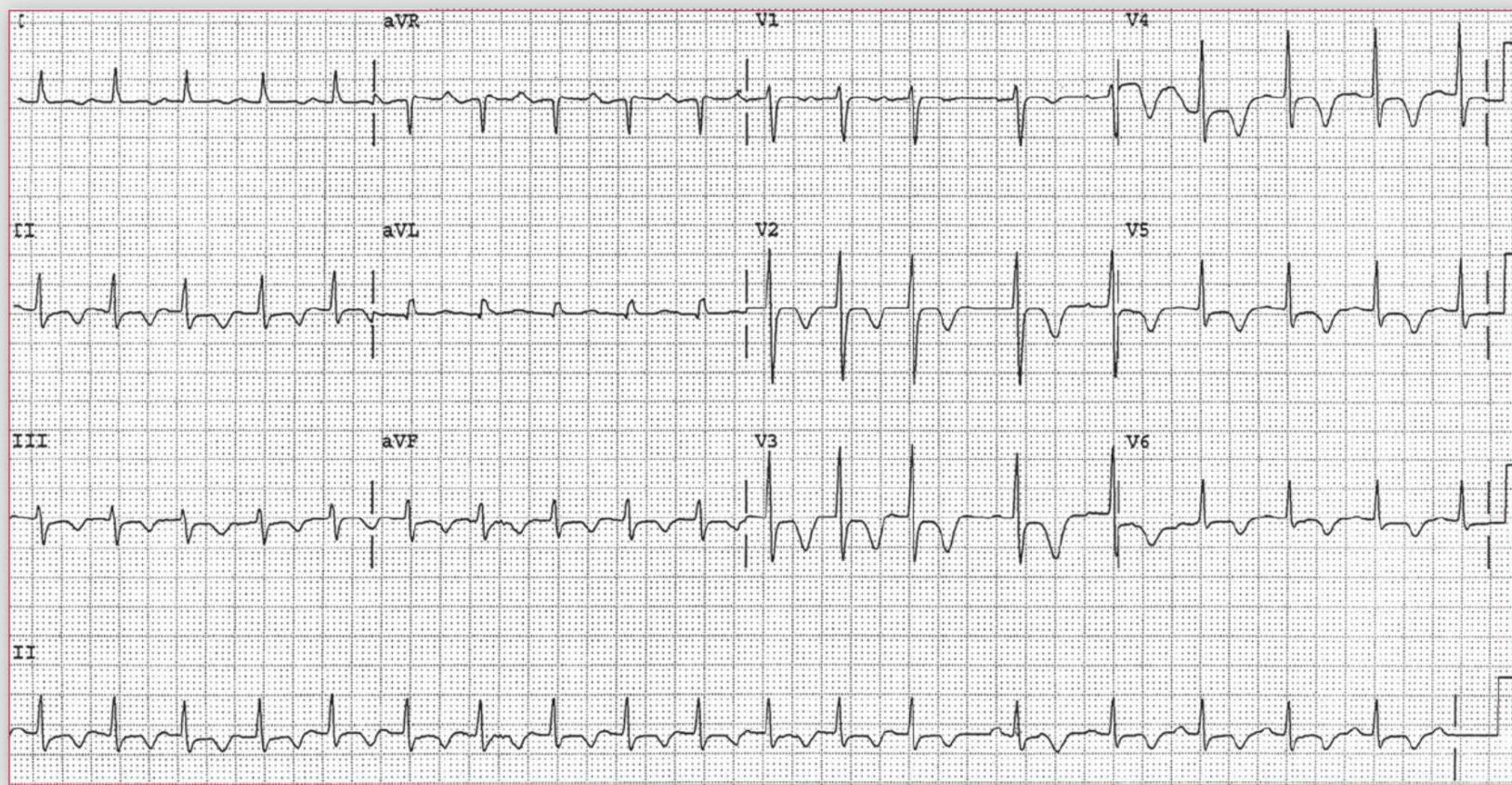
Other less common mechanisms for atrial tachycardia include reentry within a small or micro-reentrant circuit or triggered activity. When the mechanism is an ectopic focus, atrial tachycardia may result from sympathetic stimulation or an increase in circulating catecholamines. This is suggested as the etiology in this case because the arrhythmia responded to a β -blocker. However, atrial tachycardia may also result from an increase in automaticity due to atrial stretch (as occurs in heart failure), drugs such as cocaine or sympathomimetic agents, myocardial infarction, pulmonary decompensation, infection, or alcohol excess. Digoxin may provoke atrial tachycardia, possibly as a result of triggered activity, while hypokalemia and hypoxia may activate a micro-reentrant circuit. ■

Notes

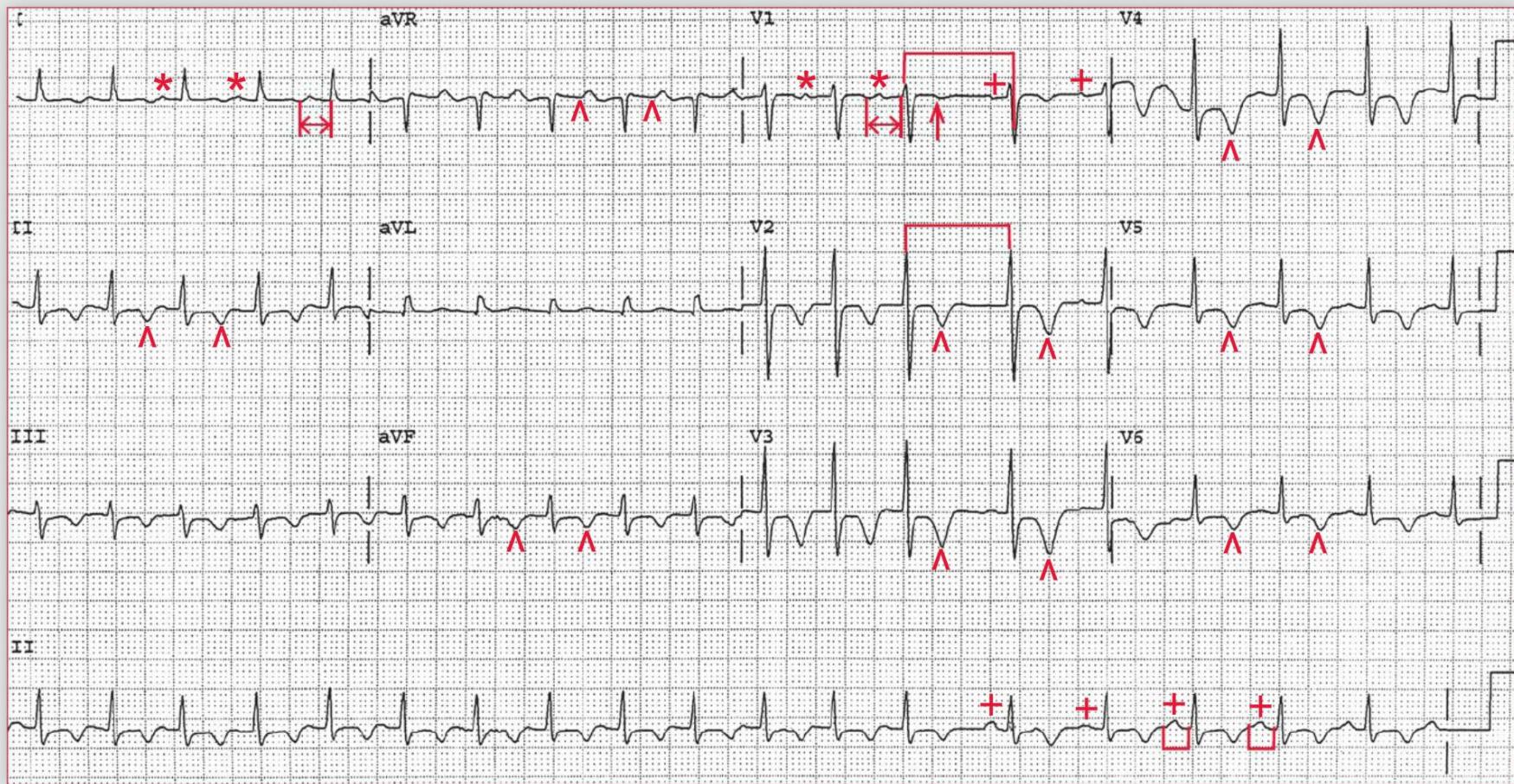
A 42-year-old man is referred to a cardiologist for progressive dyspnea on exertion and pedal edema. On echocardiogram he is found to have an ejection fraction of 38% with a moderately dilated left ventricle. There are no focal wall motion abnormalities. The cardiologist reviews the patient's ECGs from prior primary care office visits and notes that the patient is consistently tachycardic. An ECG obtained in the cardiologist's office is shown.

What does the ECG show?

What could be the cause of his cardiomyopathy?



Podrid's Real-World ECGs



ECG 16 Analysis: Ectopic atrial tachycardia terminating to a sinus rhythm, nonspecific T-wave abnormalities

The first part of this ECG shows a regular narrow complex tachycardia at a rate of 130 bpm. In most leads, there are no obvious P waves, although a P wave (*) can be seen in lead I as well as in lead V1. Of particular importance is the fact that the P wave in lead V1 is distinct. Using the PR interval established in this lead, it can be seen that the waveform in lead I is indeed the P wave. It appears to be a long RP tachycardia (*ie*, the RP interval is longer than the PR interval). The PR interval (↔) is constant (0.20 sec), and the RP interval is constant (0.28 sec). There is an abrupt slowing (◻) of the rate to 100 bpm with a P wave (+) seen before each QRS complex and a stable PR interval (◻) (0.16 sec). It can be noted in lead V1 that the P waves prior to the abrupt slowing are different than those after the pause. In addition, the PR interval with the faster rate is longer (0.20 sec) than the PR interval when the rate is slower (0.16 sec). This eliminates sinus tachycardia as the mechanism because sinus tachycardia is the result of sympathetic stimulation, which causes an increase in AV nodal conduction velocity and hence a decrease in the PR interval. The fact that the PR interval is longer during the faster heart rate means that this could not be sinus tachycardia. In contrast, the longer PR interval with

a faster rate establishes atrial tachycardia as the etiology. Therefore, the initial rhythm is atrial tachycardia that abruptly terminates to a sinus rhythm (◻). It was fortuitous that the termination of the arrhythmia was recorded on the ECG. It should be noted that the arrhythmia terminates with the absence of atrial activity (↑), indicating that the atrial activation stops abruptly. This is the way atrial arrhythmias terminate, establishing the rhythm as an ectopic atrial tachycardia.

The QRS complexes have a normal duration and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/410 msec). There are diffuse, nonspecific T-wave inversions (Λ).

If left untreated for a prolonged period of time (*ie*, weeks to months), sustained atrial tachycardia can lead to a tachycardia-mediated cardiomyopathy. This cardiomyopathy is often reversible with treatment to suppress the arrhythmia. Therapy includes a class IA, IC, or III antiarrhythmic agent or radiofrequency ablation. ■

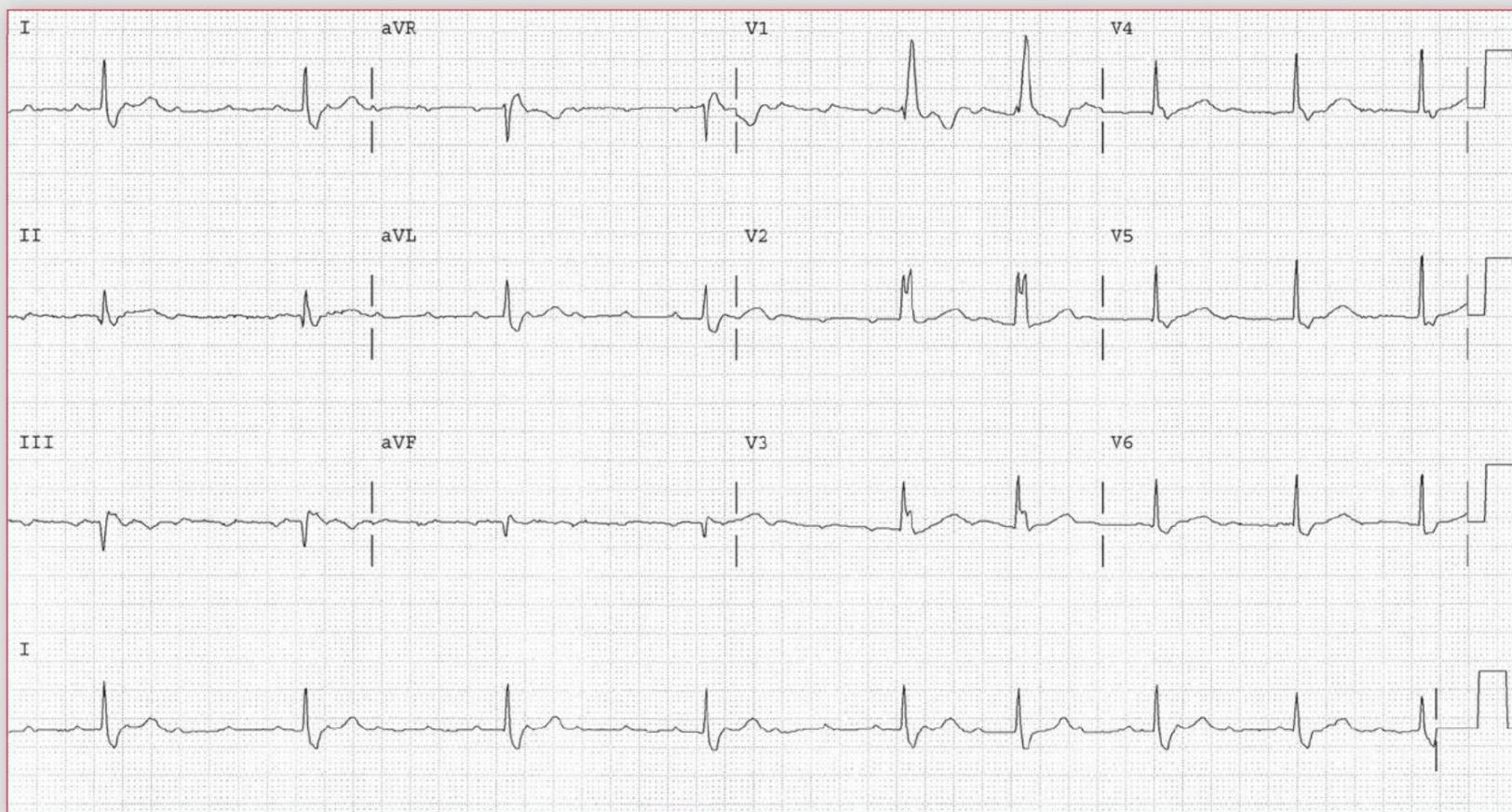
Notes

A 66-year-old woman with a prior myocardial infarction is admitted to the hospital with acute dyspnea and chest pain occurring after an international flight. She is found to have a pulmonary embolism and is started on anticoagulation. On hospital day 3, she is found to have an irregular heart rate. Her ECG is shown.

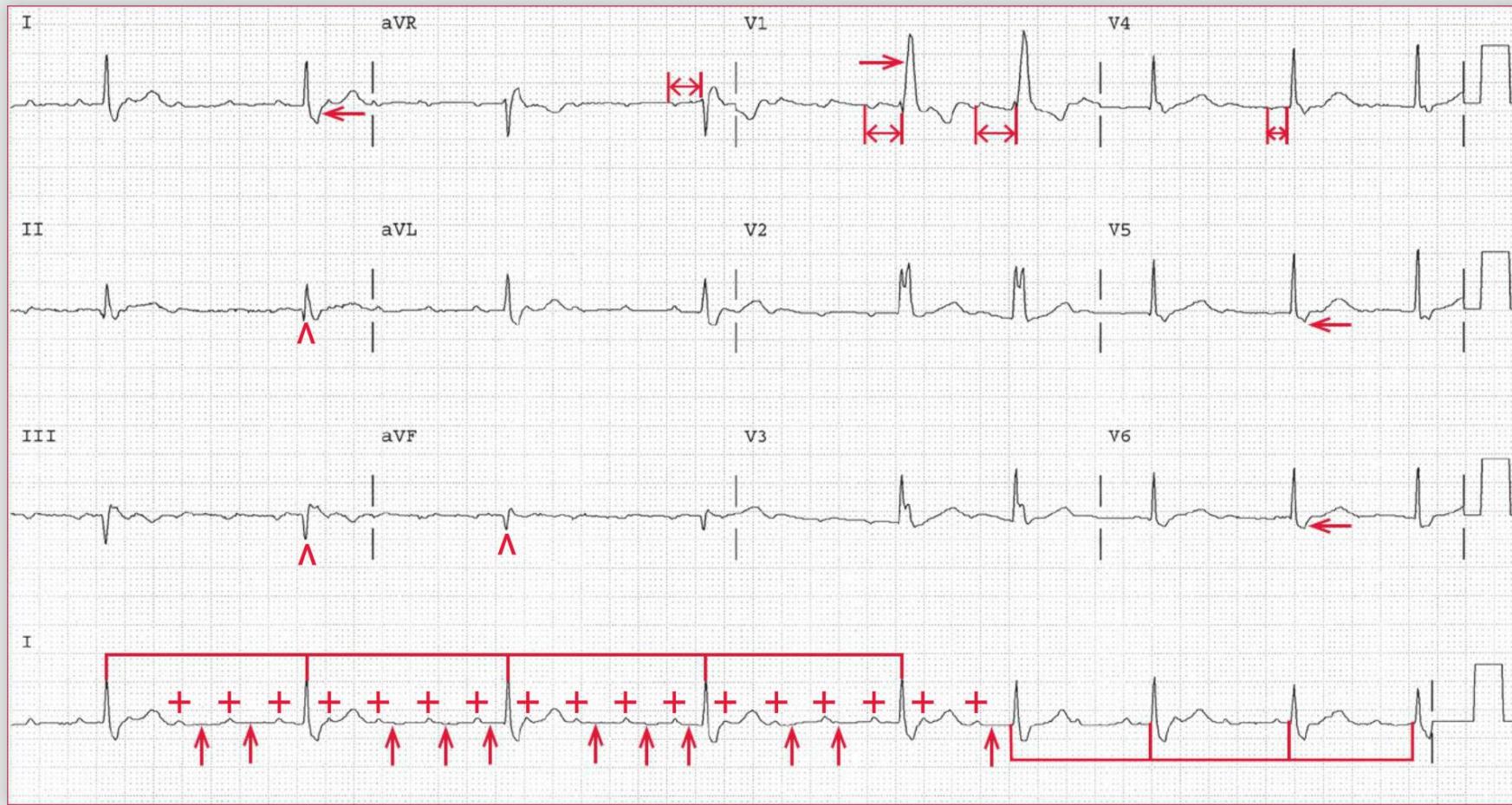
What does the ECG show?

What is the etiology?

Is any therapy necessary?



Podrid's Real-World ECGs



ECG 17 Analysis: Atrial tachycardia with variable AV block, chronic inferior wall myocardial infarction, right bundle branch block, left axis

The rhythm is irregular, but there is a pattern to the irregularity: All the long RR intervals (◻) are the same, and all the intermediate RR intervals (◻) are the same. There is an underlying atrial rate of 180 bpm, while the average ventricular rate is 56 bpm. Distinct P waves (+) are seen, and there is an isoelectric baseline (↑) between the P waves. The P waves are negative (inverted) in leads II, aVF, and V4-V6. This is, therefore, atrial tachycardia. Atrial tachycardia can be precipitated by many factors, including acute pulmonary disease. In this case, the pulmonary embolism may be the trigger for the arrhythmia.

There is variable AV conduction. Initially there is 4:1 AV conduction, then 2:1 AV conduction, and finally 3:1 AV conduction. Noted is variability of the PR intervals (↔) as a result of concealed conduction. Some atrial impulses conduct through the AV node, resulting in a QRS complex. Other impulses fail to get through the AV node (causing AV block), while still others partially penetrate the AV node (concealed). By altering AV nodal refractoriness (*ie*, partial depolarization causes an increase in AV nodal refractoriness), the concealed impulses will alter the conduction velocity through the node of the subsequent atrial depolarization, causing it to be slower.

The QRS interval is prolonged (0.16 sec), and the QRS complex morphology is typical for a right bundle branch block (RBBB) (RSR' morphology in lead V1 [→] and broad S waves in leads I and V5-V6 [←]). The RBBB may be at baseline for the patient, but may also be attributed to the pulmonary embolism because acute right ventricular pressure overload can cause an RBBB. However, this can be established by comparison with a previous ECG. The axis is leftward, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). This is a physiologic left axis. The left axis is the result of Q waves (Λ) in leads II, III, and aVF, consistent with an old inferior wall myocardial infarction. The QT/QTc intervals are normal (520/500 msec or 440/430 msec after correcting for the prolonged QRS complex duration).

The first approach in the treatment of atrial tachycardia is slowing of the ventricular rate. However, in this case, there is high-degree AV block and the ventricular rate is slow. Therefore, no acute therapy is needed. Long-term therapy for the atrial tachycardia, if it continues after resolution of the acute changes from the pulmonary embolism, would be pharmacologic therapy with a class IA, IC, or III anti-arrhythmic drug. Non-pharmacologic therapy would involve radiofrequency catheter ablation. ■

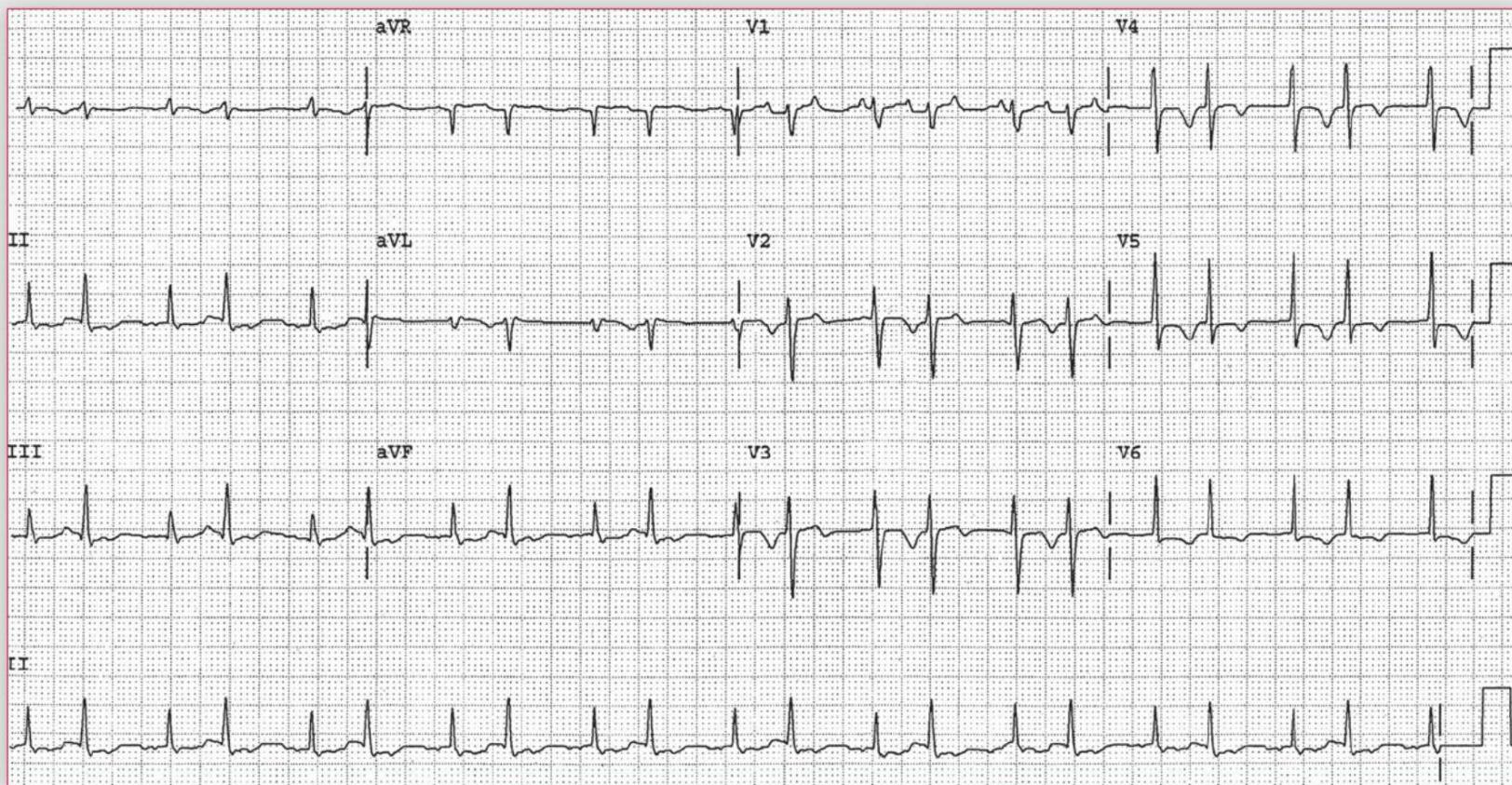
Notes

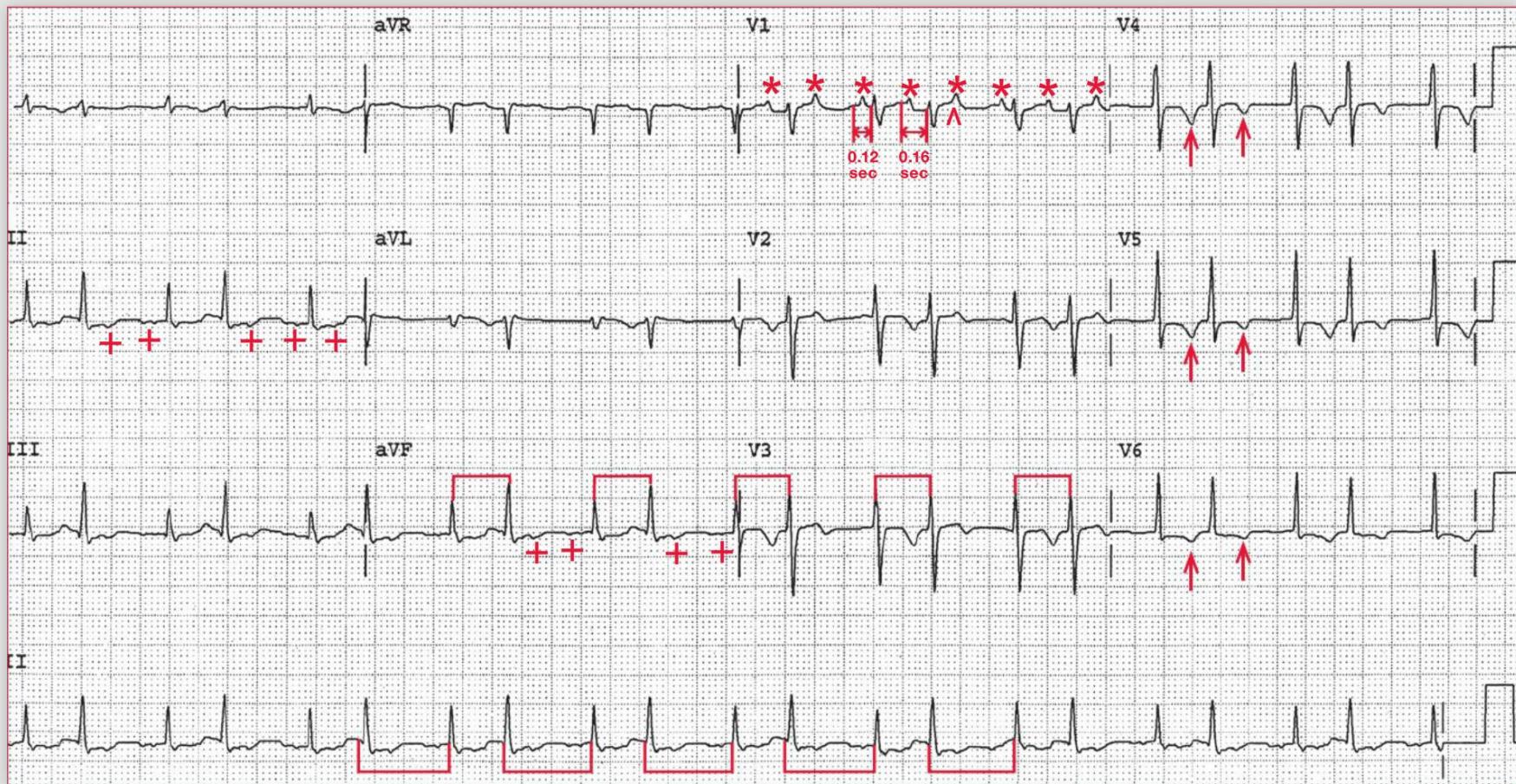
A 74-year-old man is admitted to the neurosurgical intensive care unit after undergoing a craniotomy for massive intracranial hemorrhage. He is noted to have an irregular heart rate, and an ECG is obtained.

What does the ECG show?

What is the most likely cause for the T-wave abnormalities?

What therapy would be indicated?





ECG 18 Analysis: Atrial tachycardia with 3:2 Wenckebach, diffuse T-wave inversions

The rhythm is irregular, but there is a repeating pattern of long (◻) and short (□) RR intervals (*ie*, grouped beating). The average ventricular rate is 126 bpm. Regular atrial activity can be seen, primarily in lead V1 (*), and the atrial rate is 190 bpm. The P waves are small in the other leads but appear to be negative (inverted) in leads II and aVF (+). This is atrial tachycardia. As observed in lead V1, there is a repeating pattern. The first complex of the group is associated with a short PR interval (↔) (0.12 sec) that lengthens (0.16 sec) before the second QRS complex and is followed by a nonconducted P wave (Λ). This is a pattern of 3:2 Wenckebach.

The QRS complexes have a normal duration (0.08 sec) and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/410 msec).

There are also diffuse T-wave inversions (↑) that are nonspecific but may, in this case, possibly be attributed to the intracranial bleed.

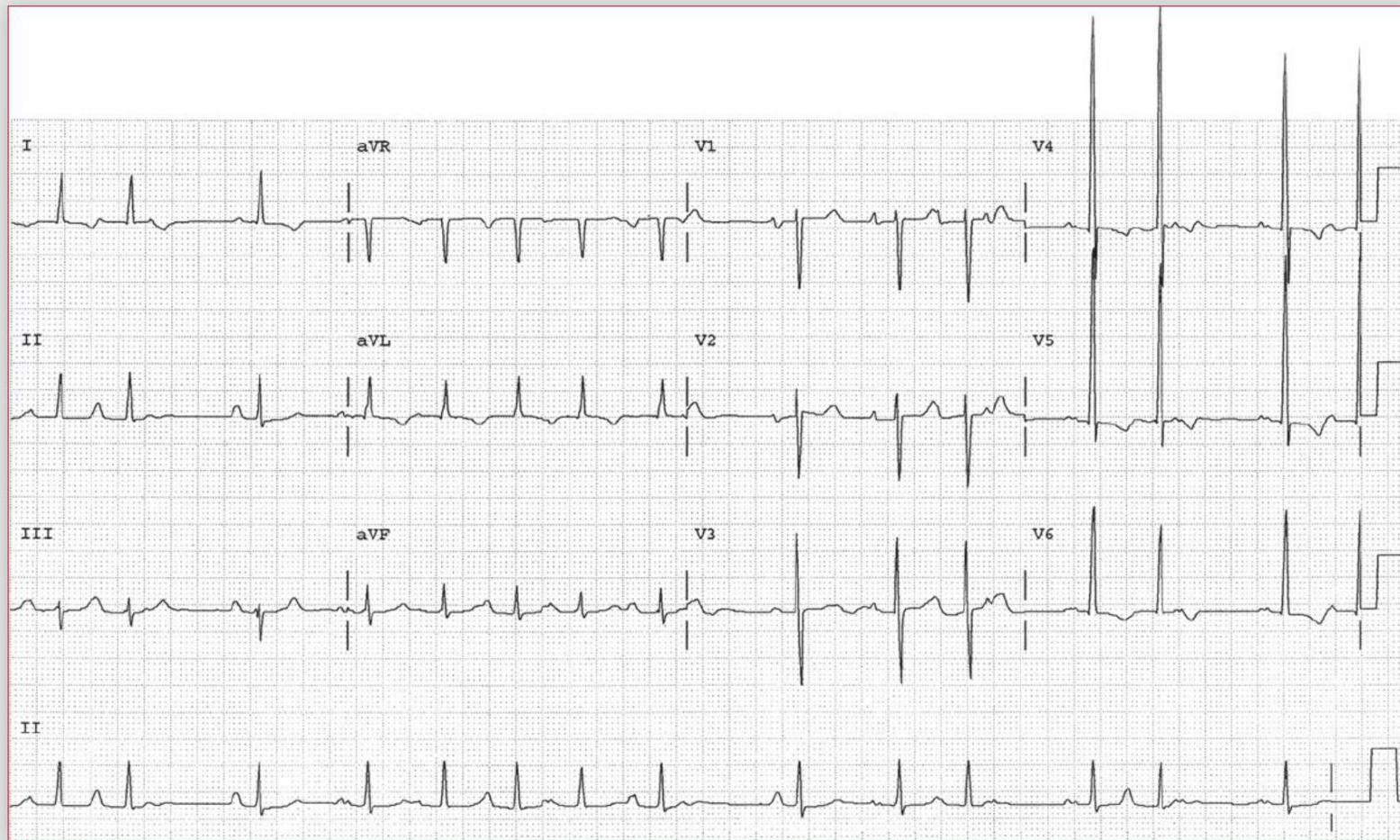
The initial therapy for atrial tachycardia is slowing of the ventricular response rate. In this case, the ventricular rate is slightly rapid and hence an AV nodal blocking agent (β -blocker, calcium-channel blocker, or digoxin) would be indicated for further rate slowing. If the arrhythmia persists after resolution of the acute neurologic problem, long-term therapy would involve a class IA, IC, or III anti-arrhythmic agent or radiofrequency catheter ablation. ■

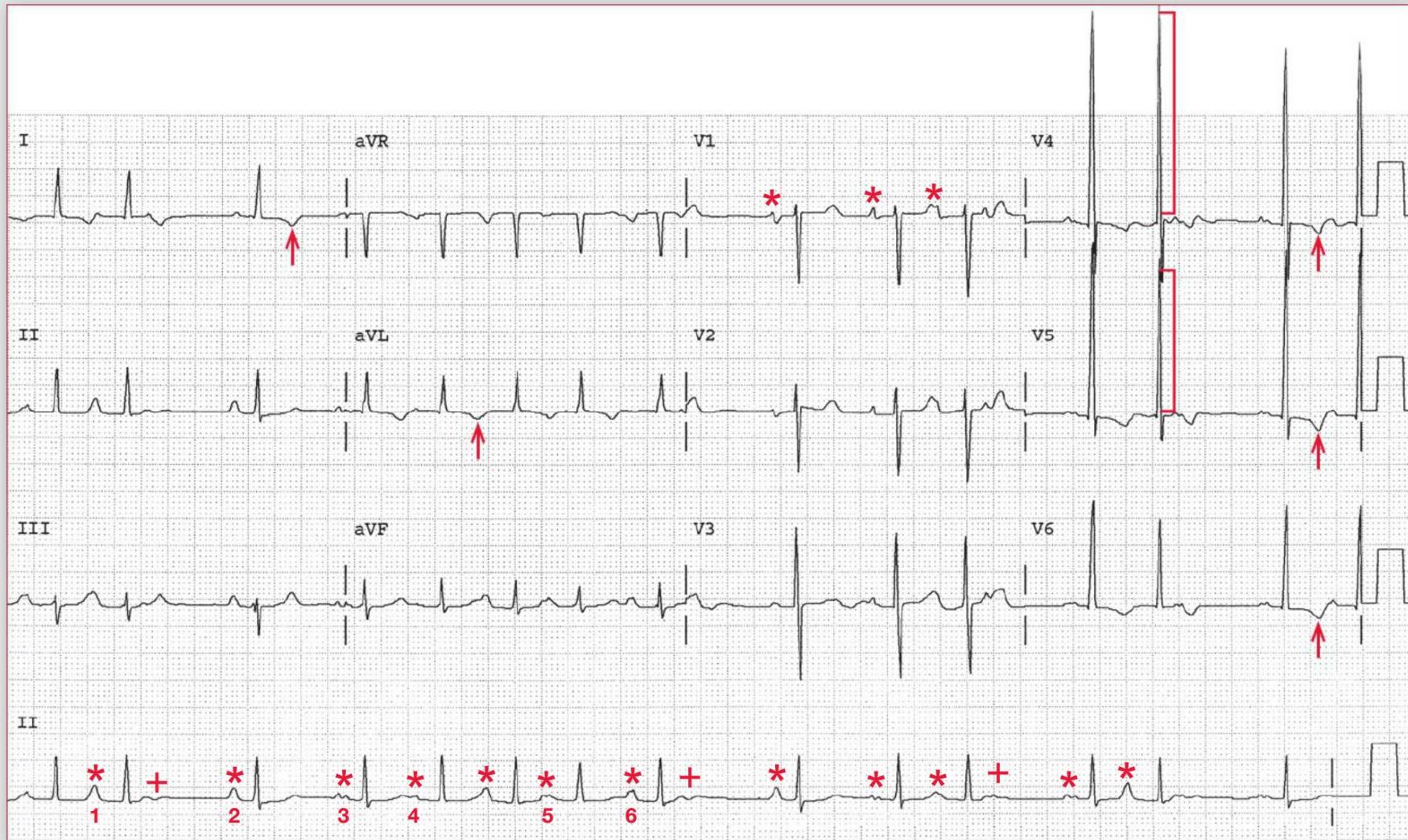
Notes

A 70-year-old man with severe chronic obstructive pulmonary disease is seen in the medical walk-in clinic for increasing cough. The nurse practitioner takes his pulse and notes an irregular heart rate. He obtains an ECG.

What does the ECG show?

What therapy is necessary?





ECG 19 Analysis: Wandering atrial pacemaker (multifocal atrial rhythm), left ventricular hypertrophy, ST-T wave changes

The rhythm is irregularly irregular at an average rate of 90 bpm. There is a P wave (*) before each QRS complex; some P waves after the QRS complexes are nonconducted (+). There are more than three different P-wave morphologies (1–6) and PR intervals. No one P-wave morphology is dominant. This is termed wandering atrial pacemaker or multifocal atrial rhythm. This arrhythmia is commonly seen in patients with chronic lung disease. When the rate exceeds 100 bpm, this is termed multifocal atrial tachycardia. It is one of three supraventricular rhythms that are irregularly irregular, the other two being atrial fibrillation and sinus arrhythmia.

The QRS complex has a normal duration (0.08 sec) and a normal axis, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/440 msec). The QRS complex amplitude in leads V4-V5 is very high (30 mm) (]), consistent with left ventricular hypertrophy. Associated with the hypertrophy are ST-T wave changes (\uparrow) in leads I, aVL, and V4-V6; these changes are due to chronic subendocardial ischemia.

Wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) is identified by the presence of a distinct P wave before each QRS complex and more than three different P-wave

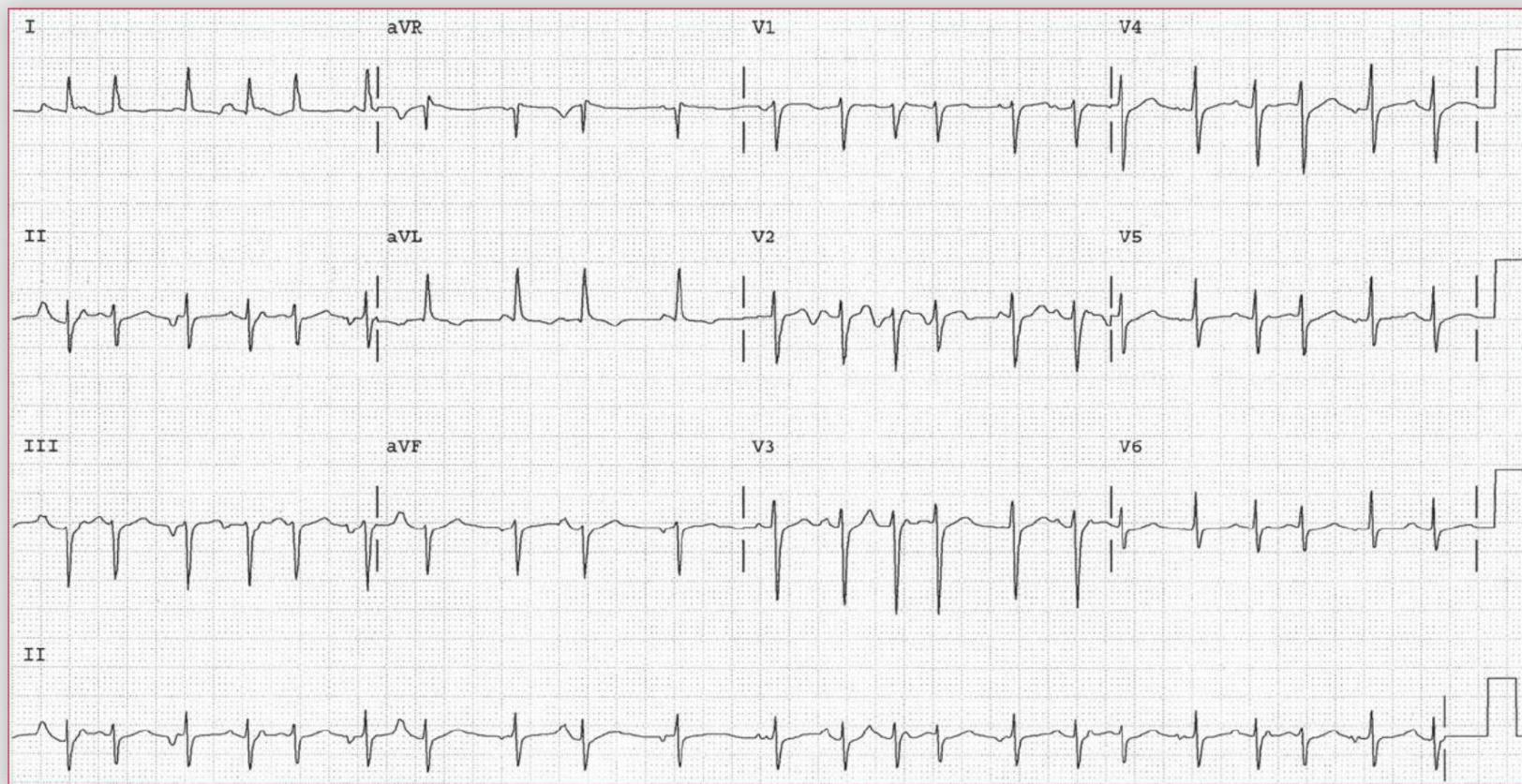
morphologies, without any one P-wave morphology being dominant. PR intervals are variable, and hence the PP and RR intervals are irregularly irregular. This arrhythmia is most commonly associated with pulmonary disease, especially when there is respiratory decompensation. Wandering atrial pacemaker (multifocal atrial rhythm) can occur in the presence of coronary, valvular, hypertensive, and other types of heart disease, particularly when associated with heart failure and pulmonary congestion or underlying lung disease. The arrhythmia often resolves spontaneously when the underlying pulmonary or cardiac problem is corrected. On occasion these arrhythmias may degenerate into atrial fibrillation. The changing morphology of the P waves and the PR interval has suggested that the pacemaker arises in different atrial locations.

Generally, no therapy is necessary for the arrhythmia itself. Rather, therapy should be directed at the underlying medical condition. If the ventricular rate is rapid, it can be slowed by blocking the AV node with a calcium-channel blocker. A β -blocker is often contraindicated if there is an underlying pulmonary problem, particularly in the presence of wheezing. There is some evidence that in the presence of hypomagnesemia or hypokalemia, magnesium or potassium replacement may be of benefit for reverting this arrhythmia. ■

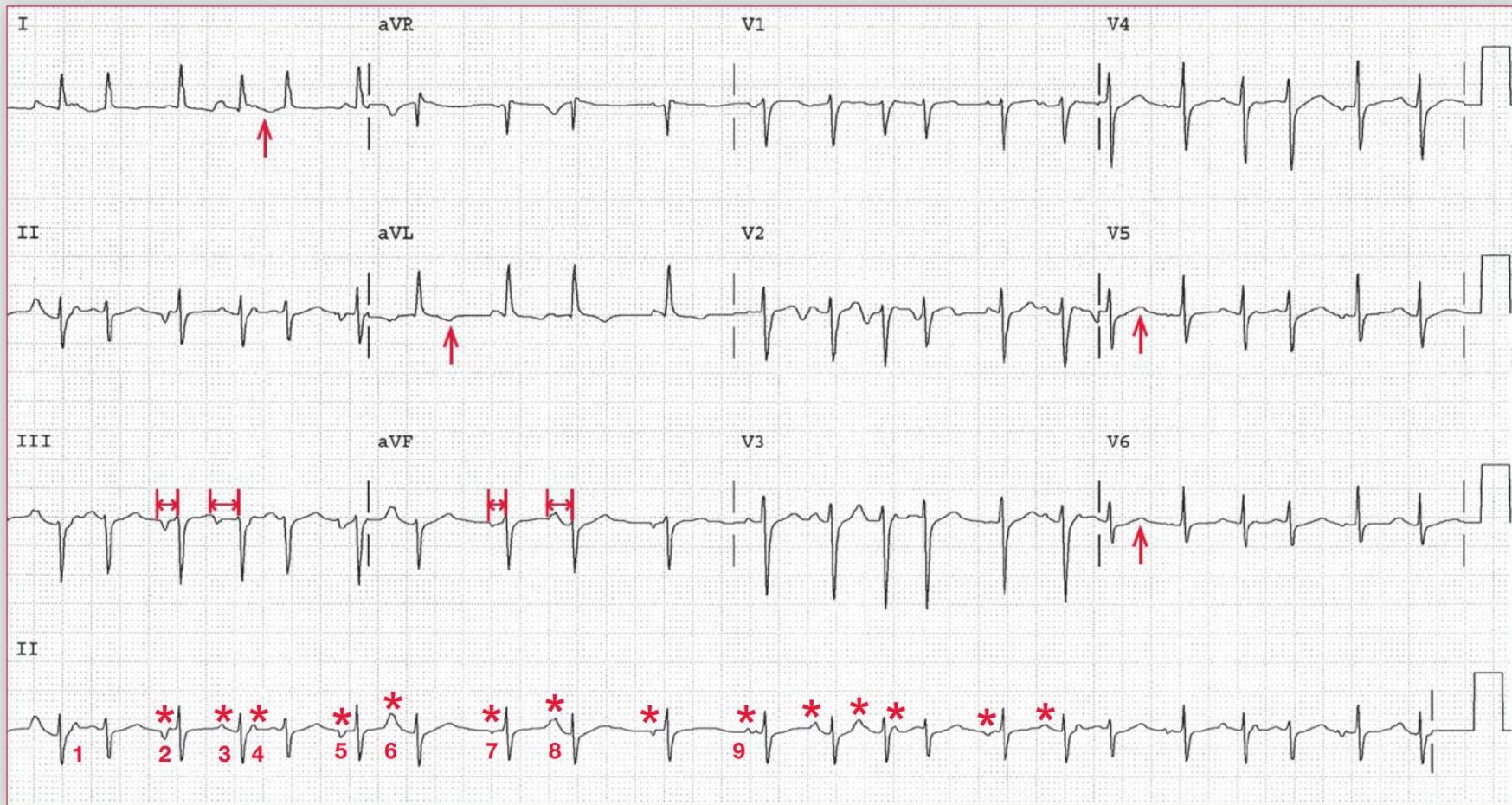
Notes

A 60-year-old man with amyotrophic lateral sclerosis is admitted to the hospital with confusion and somnolence. He is taking shallow, rapid breaths. Arterial blood gas measurements include a pCO_2 of 84 mm Hg and a pH of 7.18. He is intubated, and an ECG is obtained.

**What does the ECG show?
Is any therapy necessary?**



Podrid's Real-World ECGs



ECG 20 Analysis: Multifocal atrial tachycardia, left anterior fascicular block, nonspecific ST-T wave abnormalities

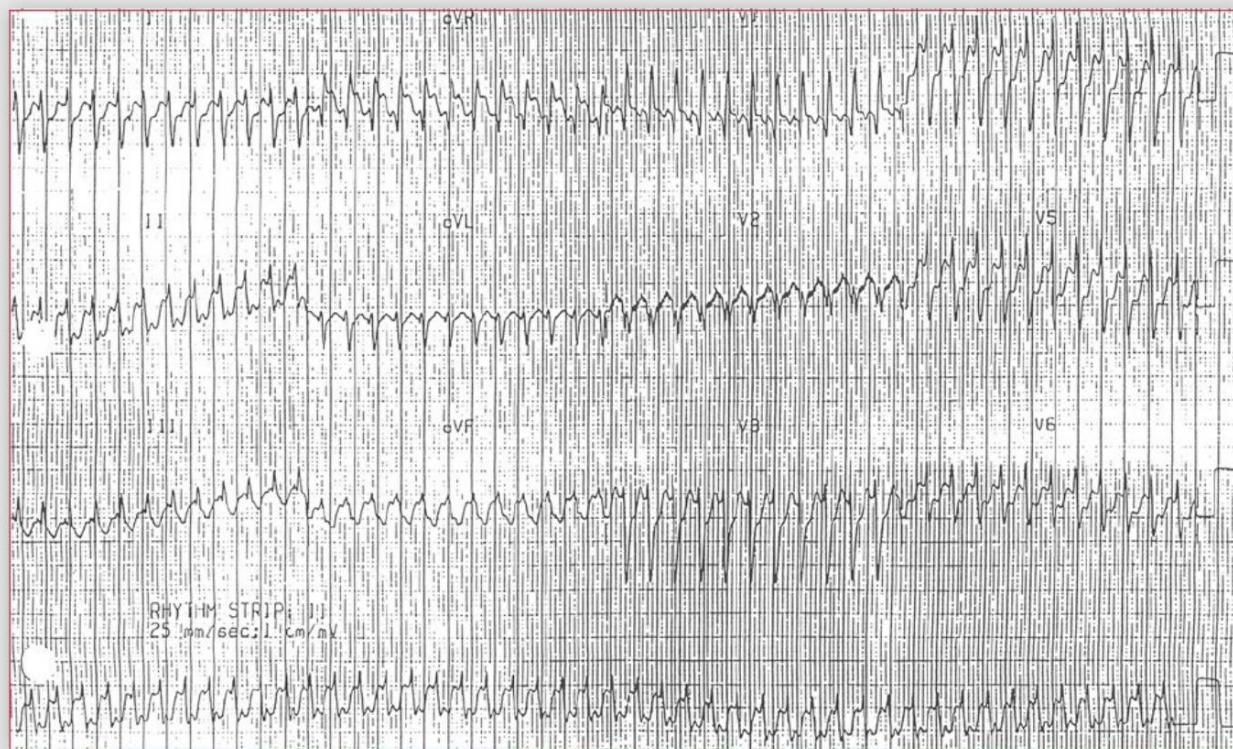
The rhythm is irregularly irregular at an average rate of 132 bpm. Although there is a P wave (*) before each QRS complex, the P-wave morphology is variable (three or more P-wave morphologies [1–6] without any single P-wave morphology being dominant). The PR intervals (↔) are not constant. Hence, this is multifocal atrial tachycardia. The QRS complexes are of normal duration (0.08 sec) and morphology. The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology), hence this is a left anterior fascicular block. The QT/QTc intervals are normal (280/420 msec). Diffuse ST-T wave abnormalities (↑) can be seen.

Multifocal atrial tachycardia is an arrhythmia that occurs in a variety of situations, most commonly congestive heart failure and pulmonary congestion or underlying lung disease. Affected patients tend to have elevated pulmonary capillary wedge and pulmonary end-diastolic pressures as well as a low-normal cardiac index. Other factors can also predispose to this arrhythmia, including autonomic imbalance, hypercarbia, and acidosis. Therapy for the arrhythmia in this case involves treatment of the underlying medical problem. If the ventricular rate is rapid and associated with symptoms, ventricular rate control can be achieved with AV nodal blocking agents (*ie*, digoxin, calcium-channel blockers, β -blockers). ■

Core Case 21

A 26-year-old man presents to the emergency department after noting the acute onset of palpitations and lightheadedness while self-administering an albuterol nebulizer at home for a known diagnosis of asthma. He has no other medical conditions and takes no medications other than his bronchodilator. On exam, his heart rate is approximately 300 bpm and his blood

ECG 21A



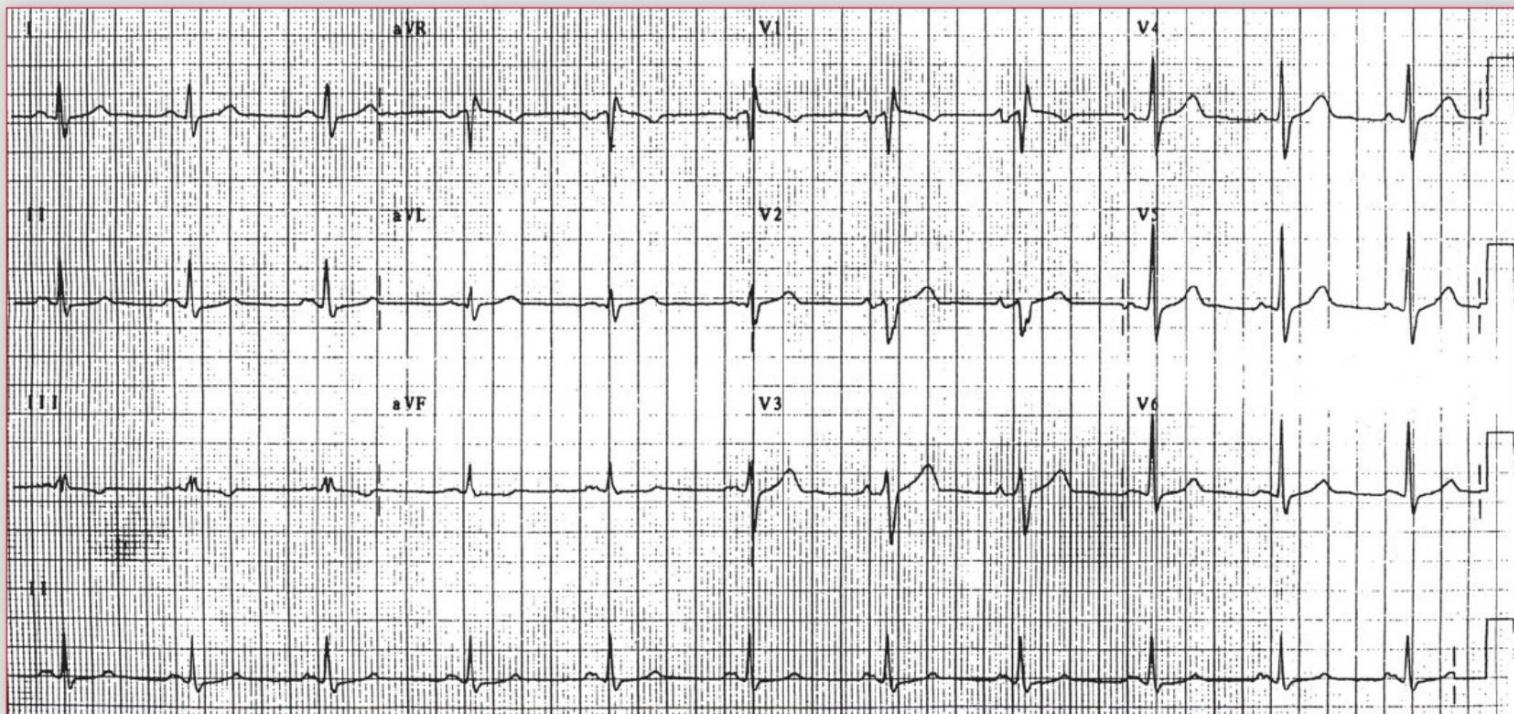
pressure is 108/74 mm Hg. He is in mild distress and slightly tachypneic. His pulse is rapid and regular. His cardiopulmonary exam is otherwise normal. An ECG is obtained (ECG 21A). A β -blocker is administered, and there is slowing of the heart rate (ECG 21B). The ECGs are compared with the patient's baseline ECG (21C).

ECG 21B



Core Case 21

ECG 21C



What is the diagnosis?

What is an appropriate next clinical maneuver?



ECG 21A Analysis: 1:1 atrial flutter, intraventricular conduction delay to the right ventricle (R' waveform in lead V1)

The rhythm in ECG 21A is regular at a rate of 300 bpm. The QRS complex duration is normal (0.10 sec). Hence this is a supraventricular rhythm. The only supraventricular arrhythmia that occurs with a regular atrial rate faster than 260 bpm is atrial flutter. As the ventricular rate is 300 bpm, this is atrial flutter with 1:1 AV conduction.

There is an RSR' morphology in lead V1 (↓) and an S wave (↑) in leads I and V5-V6; this represents a right ventricular conduction delay. This has also been called an incomplete right bundle branch block (as the QRS complex is less than 0.12 sec in duration). However, conduction through the His-Purkinje system and bundles is all or none and is not partial or incomplete. Rather, incomplete conduction is actually diffuse slowing of conduction through the His-Purkinje system (*ie*, an intraventricular conduction delay to the right ventricle).

Also noted is significant diffuse ST-segment depression (+) (ST-segment elevation in lead aVR [●] is actually ST-segment depression). Although ST-segment depression due to ischemia is possible at a rate of 300 bpm, it is likely that the apparent ST-segment depression is actually the flutter wave.

Atrial rates between 260 and 320 bpm are diagnostic for atrial flutter (type I or typical atrial flutter). At these rates the unstimulated AV node is not capable of conducting each impulse. In most cases, the AV node exhibits decremental conduction; that is, with an increasing atrial rate or increased frequency of depolarizing stimuli stimulating the AV node there is a progressive decrease in the conduction velocity of the impulse through the AV node due to a progressive increase in the refractory period. The result is the occurrence of heart block during times of rapid

atrial rates. Commonly, the QRS complex rate is an integer division of the atrial rate (2:1, 3:1, 4:1, etc.). In some cases Wenckebach block is present, often with a pattern of 3:2 conduction. Uncommonly, the atrial and ventricular rates may have a 1:1 relationship (*ie*, atrial flutter with 1:1 conduction). This may be seen when there is an increase in sympathetic inputs into the AV node or an increase in circulating catecholamines. This results in shortening of AV nodal refractoriness and enhancement of conduction velocity through the AV node. Situations in which this occurs include exercise, hyperthyroidism, infection, use of sympathomimetic drugs, and heart failure. It may be more frequent in younger patients in whom the AV node is normal.

As this patient is hemodynamically stable, appropriate therapy would include slowing AV conduction for both diagnostic and therapeutic purposes. A short-acting AV nodal blocking agent such as adenosine may be used in the acute setting for an immediate effect. However, given a half-life of only a few seconds, adenosine will do little more than provide an electrocardiographic view of the underlying atrial rhythm (*ie*, exposing the atrial waveforms) and aid in diagnosis. A longer-acting nodal agent (intravenous β -blocker or calcium-channel blocker) must be used for a more long-lasting clinical effect. If there is hemodynamic instability, urgent cardioversion should be used.

Once stable, a search for causes of atrial flutter in this young patient must be initiated. Common causes in the general ambulatory patient include thyrotoxicosis, pericardial disease, pulmonary embolism, mitral valve disease, and sick sinus syndrome; atrial flutter may also be idiopathic.

continues



ECG 21B Analysis: Atrial flutter with variable AV conduction (variable AV block),
intraventricular conduction delay to the right ventricle (R' waveform in lead V1)

A second ECG (21B), obtained after administration of an AV nodal blocking agent (β -blocker), shows that the heart rate has slowed to 108 bpm and the rhythm is irregular. However, there is a pattern to the irregularity as a result of variable AV conduction. Hence the rhythm is regularly irregular. Regular atrial flutter waves (+) are now seen at a rate of 300 bpm. This is, therefore, atrial flutter with variable AV block or variable AV conduction.

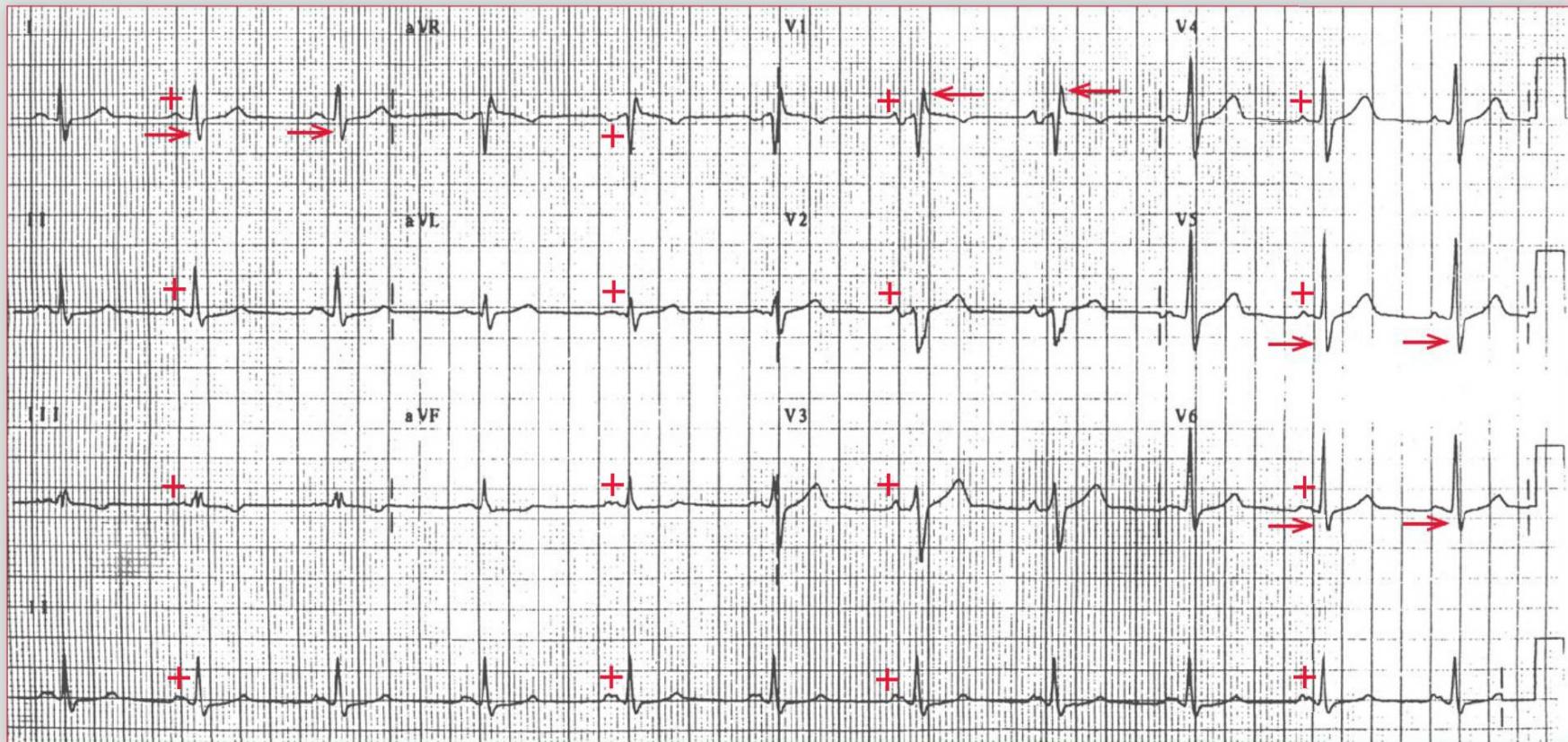
The QRS complex duration is normal (0.10 sec) with an RSR' morphology in lead V1 that represents a right ventricular intraventricular conduction delay (\leftarrow), as seen in ECG 21A. The QT/QTc intervals are normal (320/430 msec). However, ST-segment depression is no longer present, confirming the fact that the abnormality in the ST segment was the result of the superimposed flutter wave and not ST-segment depression as a result of ischemia.

The atrial rate in typical atrial flutter is 260 to 320 bpm. Flutter waves (negative/positive in leads II, III, and aVF) are uniform in morphology, amplitude, and interval. There is no isoelectric baseline between flutter

waves as they are continuously undulating, described as saw tooth. The flutter rate may be slower than 260 bpm as a result of anti-arrhythmic drugs or disease of the atrial myocardium; however, the atrial waveforms maintain the typical flutter morphology.

QRS complex intervals are regular when there is a repeating pattern of AV block (*eg*, 2:1, 3:1, 4:1, etc). However, the rhythm may be regularly irregular when there is variable block or if Wenckebach is present. The RR intervals are related to the underlying atrial rate, and hence the rhythm is regularly irregular. In addition, there may be slight irregularity of the RR interval related to a variable relationship between the flutter wave and the QRS complex (\leftrightarrow) due to antegrade concealed AV nodal conduction. In this situation some of the atrial impulses conduct through the AV node to activate the ventricles, some impulses are blocked within the AV node, and other impulses only partially penetrate and depolarize the AV node (*ie*, they are concealed within the AV node). However, the partial depolarization and partial refractoriness of the AV node result in a slowing of AV nodal conduction of the subsequent impulse.

continues



ECG 21C Analysis: Normal sinus rhythm, intraventricular conduction delay to the right ventricle (R' waveform in lead V1)

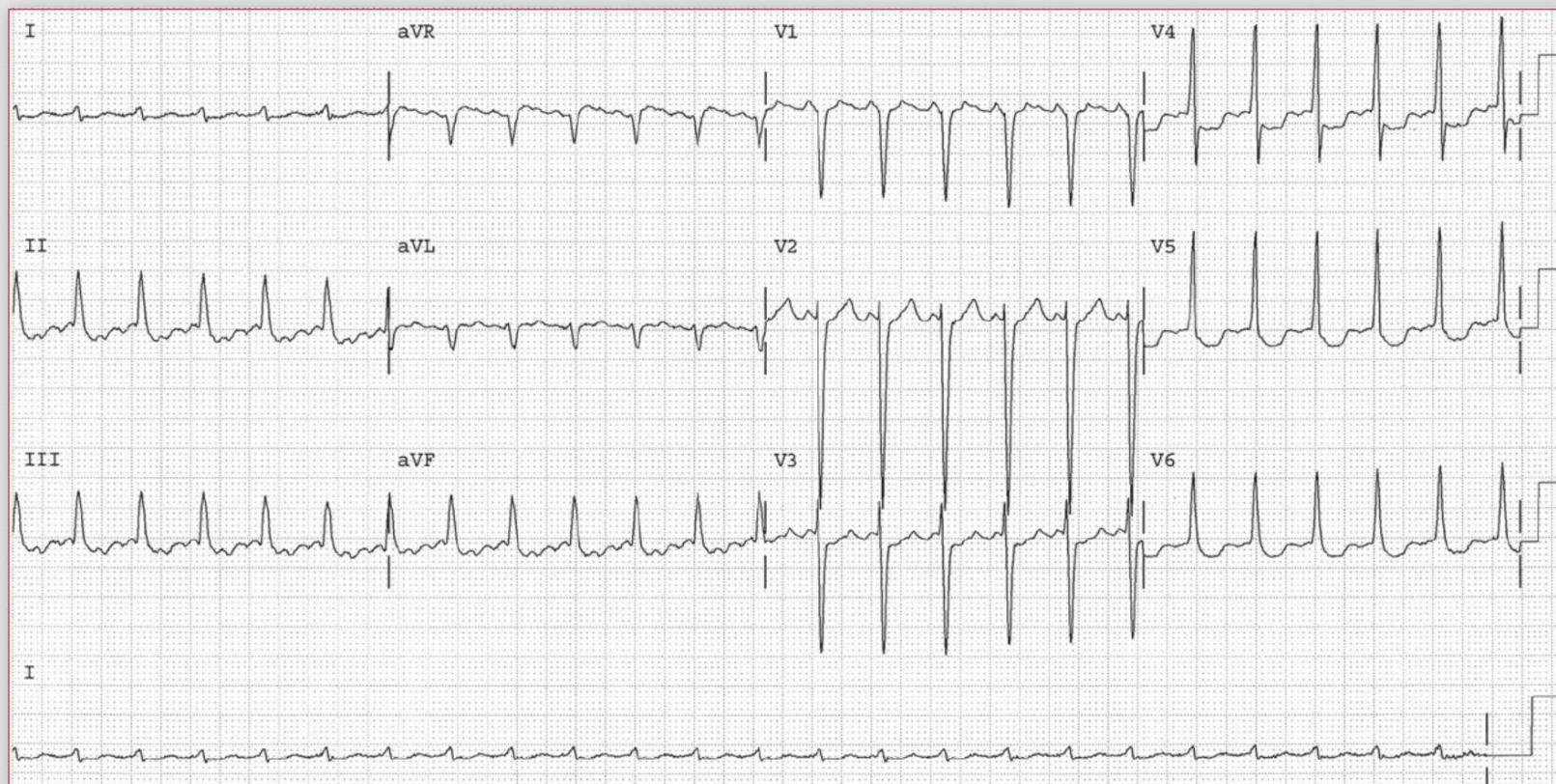
In ECG 21C the rhythm is regular at a rate of 66 bpm. There is a P wave (+) (with a normal morphology) in front of each QRS complex with a stable PR interval. The P wave is positive in leads I, II, aVF, and V4-V6. This is, therefore, a normal sinus rhythm. The QRS complex duration is normal (0.10 sec), and there is an RSR' morphology in lead V1 (←) and a prominent S wave in leads I and V5-V6 (→), a result of a right ventricular intraventricular conduction delay. The QRS complex morphology is similar to that seen in ECGs 21A and 21B. The QT/QTc intervals are normal (400/420 msec). ■

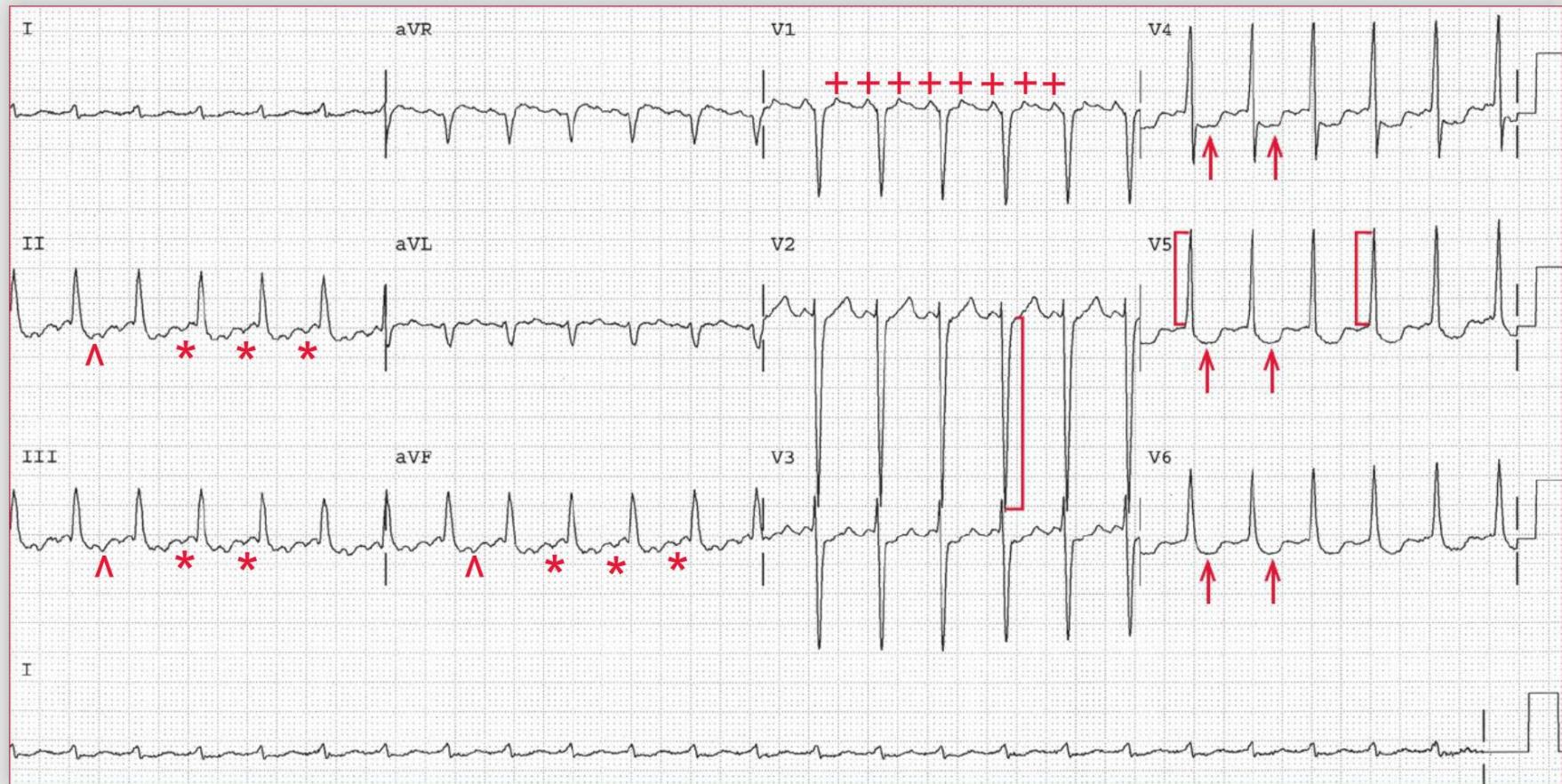
Notes

A 57-year-old woman with a long history of untreated hypertension presents to an urgent care clinic with complaints of 1 day of palpitations. She denies any associated symptoms, specifically dyspnea, diaphoresis, chest pain, or anxiety. The symptoms are constant. She does not take medications and

denies recent stimulant use. On exam, her pulse is rapid and regular. Her blood pressure is 168/94 mm Hg. Her jugular venous pressure is normal with normal descents. Her lungs are clear, and her cardiac exam is without lifts, murmurs, or rubs. The remainder of her exam is unremarkable. You obtain an ECG.

What is the diagnosis?





ECG 22 Analysis: Atrial flutter with 2:1 conduction (2:1 AV block),
left ventricular hypertrophy with ST-segment depression

There is a regular rhythm at a rate of 150 bpm. Because the most common rate of atrial flutter is 300 bpm, atrial flutter with 2:1 AV conduction must be considered whenever there is regular supraventricular tachycardia at a rate of 150 bpm. Distinct negative atrial waveforms can be seen in leads II, III, and aVF (*). Looking more closely at lead V1, two distinct positive atrial waveforms can be seen (+), one just before the QRS complex and the second superimposed on the T wave. On closer inspection the second flutter wave can indeed be seen after the QRS complex in leads II, III, and aVF (Λ), although initially it might have been considered an abnormal ST-T wave. There is a regular PP interval, and the atrial rate is 300 bpm. Hence the rhythm is typical atrial flutter with 2:1 AV conduction.

The QRS complexes are of normal duration (0.08 sec) and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (240/380 msec). The amplitude of the QRS complex is increased; the S-wave depth in lead V2 is 33 mm (]) and the R-wave amplitude in lead V5 is 17 mm ([) (S-wave depth in lead V2 + R-wave amplitude in lead V5 = 50 mm). The QRS complex amplitude is diagnostic for left ventricular hypertrophy. There is also ST-segment depression in leads V3-V6 (↑), which is consistent with subendocardial ischemia that may be primary as a result of the rapid rate or secondary to left ventricular hypertrophy.

Atrial flutter is a supraventricular arrhythmia with a regular atrial rate between 260 and 320 bpm, most commonly 300 bpm. There are two forms of atrial flutter based on the electrical mechanism of the arrhythmia.

Type I (or typical) atrial flutter is a result of reentry due to an anatomic block. It is also called “isthmus-dependent” atrial flutter as it involves an area of slow conduction through an isthmus of tissue in the right atrium between the vena cava and the tricuspid valve annulus, along the Eustachian ridge.

The right atrium contains numerous anatomic features that represent relative anatomic barriers to electrical conduction. Specifically the sinus venosus (region between the venae cavae) and os of the coronary sinus represent a posterior boundary. The tricuspid valve annulus represents an anterior boundary. These features define one limb of a loop around the tricuspid valve annulus within which exists the Eustachian ridge, an area of slowed electrical conduction. Impulses in this loop are forced to travel through the area of slowed conduction. They may be sufficiently slowed to allow the surrounding atrial tissue to repolarize before the impulse exits. This will result in reexcitation of atrial tissue by the impulse exiting the area of slowed conduction rather than by impulses from the sinus node, setting up a perpetual circuit of conduction around the right atrial walls. This loop-like phenomenon of electrical conduction is termed “reentry,” and specifically “macro-reentry” as it involves a large anatomic path of conduction (in contrast to micro-reentry, which involves reentry on a microscopic scale). As a result of the area of slow conduction due to the anatomic block, there is a wide or long excitable gap, which allows time for an impulse to enter the circuit (*ie*, with cardioversion) and depolarize the tissue of this gap or slow conduction, causing it to become refractory and hence interfering with the circuit and terminating the arrhythmia. Since there

continues

is a long excitable gap, this arrhythmia is easily treated with radiofrequency ablation. If a catheter with a simulating electrode is placed within this electro-anatomic loop and electrical stimulation is provided at the correct frequency, the arrhythmia can be reproduced, a maneuver referred to as “entrainment.” In addition, a stimulating electrode delivering impulses at a rate more rapid than the atrial rate can enter and capture this circuit, resulting in an increase in the atrial rate; this is also “entrainment.”

During atrial flutter, given the speed at which electrical signals travel in healthy atrial tissue, it takes approximately 200 msec for an atrial impulse to complete one peri-annular loop; therefore, the atrial flutter rate will be approximately 300 bpm (200 msec/beat) in isthmus-dependent flutter. The typical range for flutter wave periodicity is 260 to 320 bpm, but it may be slower if the patient has been exposed to drugs that decrease myocardial impulse conduction (*eg*, class IA or IC anti-arrhythmic drugs owing to their inhibition of sodium channels) or prolong atrial refractoriness (*ie*, class III anti-arrhythmic drugs that inhibit potassium channels). The atrial flutter rate may also be slower if there is underlying atrial myocardial disease and fibrosis.

Type I atrial flutter is further subdivided into clockwise and counterclockwise flutter based on the direction of travel of the reentrant circuit around the tricuspid valve annulus. The direction of travel creates a characteristic flutter-wave morphology on the surface ECG. Flutter waves are best seen in inferior leads II, III, and aVF, and in lead V1. Clockwise (or reverse) flutter is less common (approximately 10% of type I flutter cases) and is characterized by predominantly positive

flutter waves as seen in the inferior leads and negative deflection in lead V1. Counterclockwise flutter is the overwhelmingly predominant form of type I flutter and is characterized by negative-positive flutter waves as seen in the inferior leads and positive waves in lead V1.

Type II (atypical) flutter is a reentrant circuit that is not due to an anatomic block or area of slow conduction; that is, it is not isthmus-dependent. It is the result of a functional block (*ie*, an area of prolonged refractoriness that results in a small area of slow conduction). In this situation, the circuit is small and there is only a very short or narrow excitable gap. As a result the flutter rate is faster (generally 340 to 440 bpm), the flutter waves are generally positive in the inferior leads, the flutter is unable to be entrained (unlike type I, which is defined in part by its ability to be entrained about the isthmus), and it is less amenable to cardioversion or radiofrequency ablation as there is very little time or opportunity for an impulse to enter the reentrant circuit to terminate the arrhythmia (a result of the narrow excitable gap).

Atrial flutter may be a difficult rhythm to diagnose as one of the flutter waves may be within the QRS complex. It may be at the end of the QRS complex, resembling an S wave or even ST-segment depression, or at the beginning of the QRS complex, suggesting a Q wave. In the case presented, the flutter waves are within the T wave and partially hidden within the beginning of the following QRS complex. These overlaps not only make discerning the flutter waves difficult, but they make characterization of the flutter as clockwise or counterclockwise challenging as well. ■

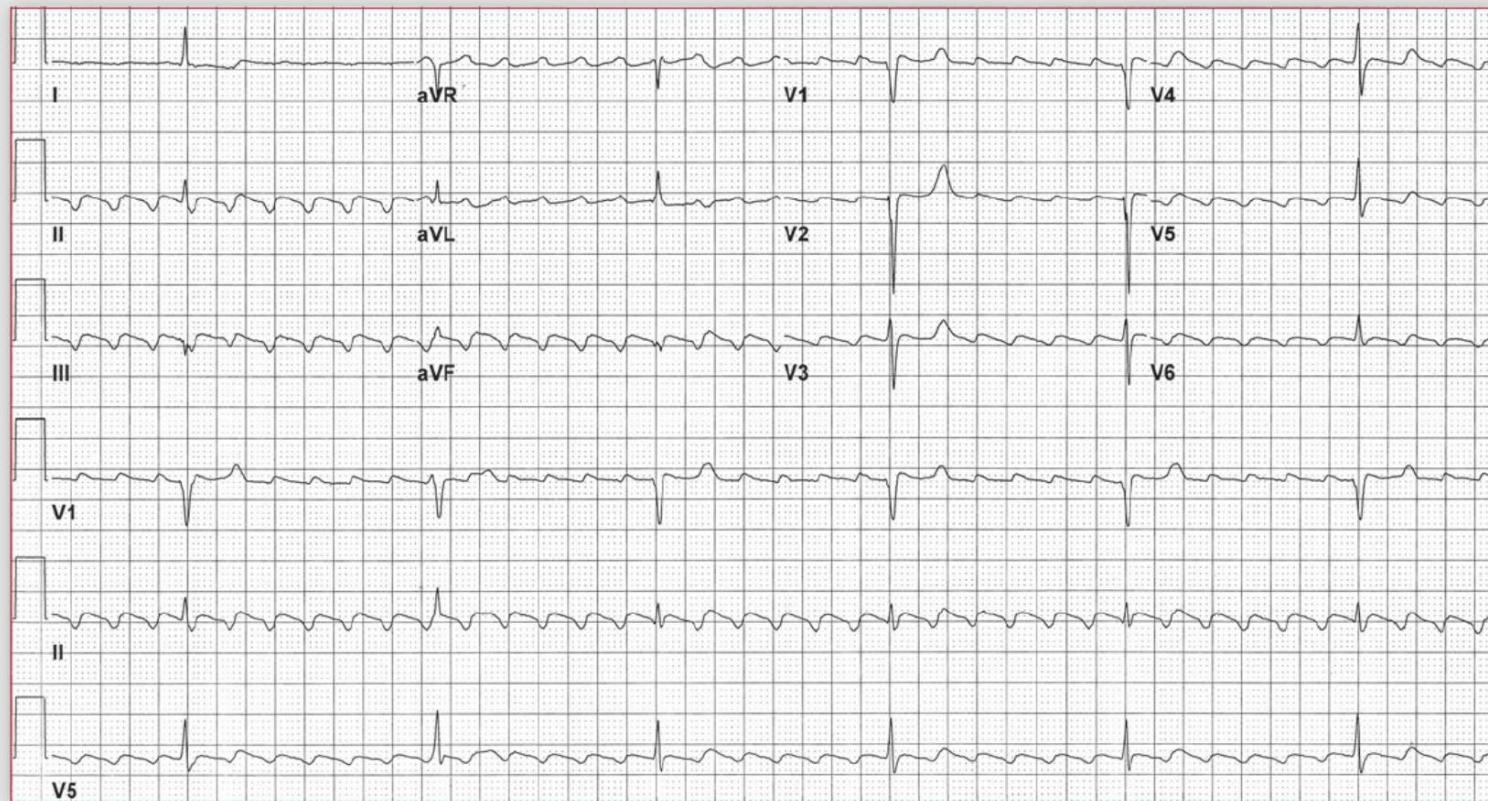
You are called to the emergency department to see a 55-year-old man who has presented with palpitations, shortness of breath, and mild chest discomfort. The emergency department physician reports that the patient has supraventricular tachycardia (SVT) and rapid ventricular conduction, observed to be at a rate of 240 bpm. She administers intravenous adenosine upon your arrival. Shown here is a 12-lead ECG taken just after adenosine infusion.

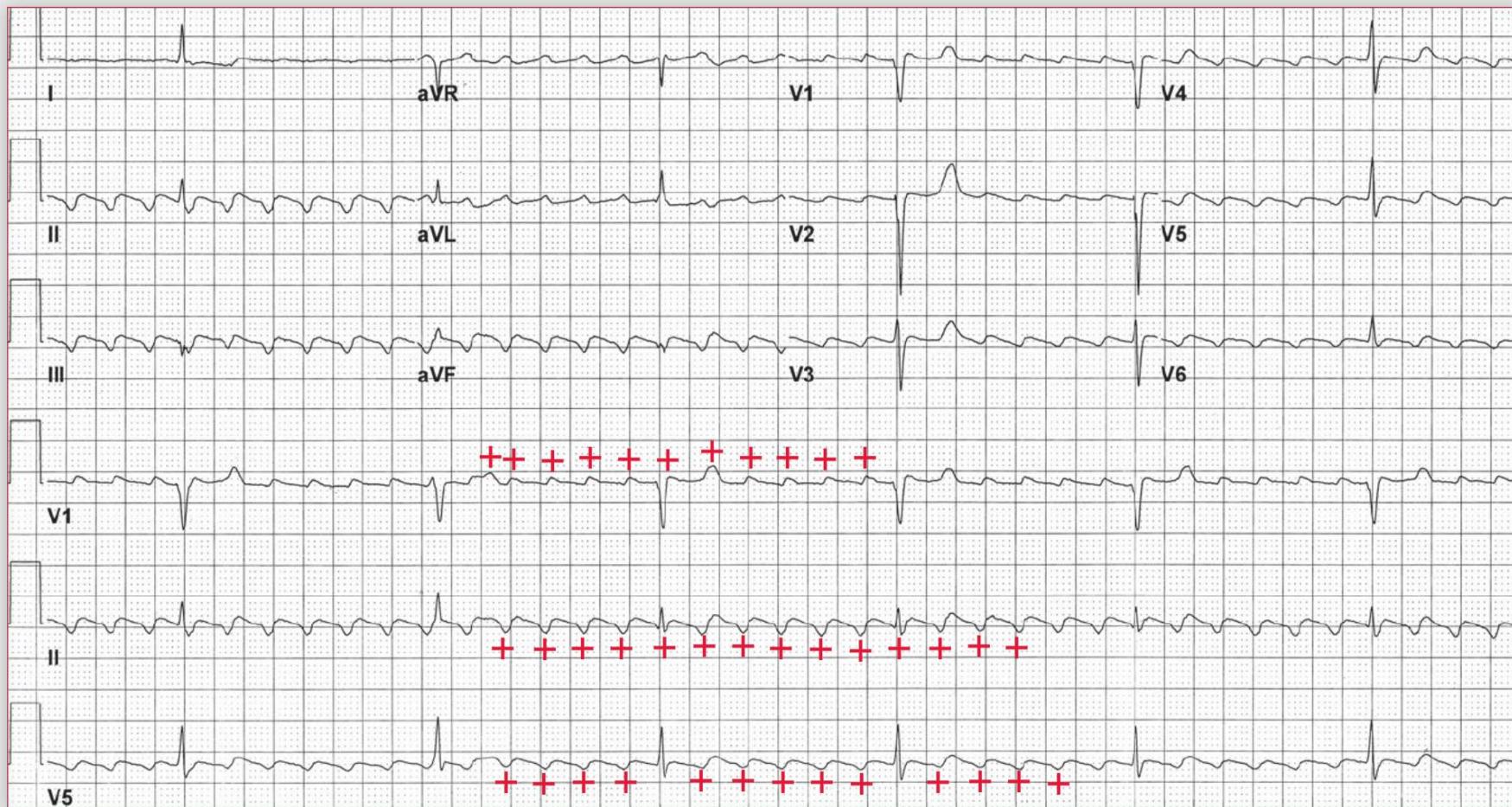
You are told that the man has a history of paroxysmal atrial fibrillation and takes flecainide for rhythm control. He has no other medical diagnoses and takes no other medications.

What is the utility of intravenous adenosine in this case, and what specific type of SVT is suggested by the ECG?

What resulted in the patient's rapid ventricular rates on presentation?

What can be done to prevent this in the future?





ECG 23 Analysis: Atrial flutter with 7:1 conduction, low-voltage limb leads, left axis, poor R-wave progression in leads V1 and V2

There is a regular rhythm at a rate of 38 bpm, with the first RR interval being slightly longer than subsequent intervals. As a result of high-degree AV block, prominent atrial flutter waves are seen (+), occurring at a rate of 240 bpm. The flutter waves are uniform in morphology, amplitude, and interval and have a typical undulating (saw-tooth) pattern. There is no isoelectric baseline between the flutter waves. The AV conduction varies, being 8:1 for the first RR interval and 7:1 for the other RR intervals.

The QRS complexes are normal in duration (0.08 sec) with a physiologic left axis, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). There is low QRS voltage in the limb leads (< 5 mm in each lead). The QT/QTc intervals are normal (440/350 msec).

Adenosine induces hyperpolarization of AV nodal tissue by interacting with the A1 receptor, thereby reducing cAMP levels and increasing potassium ion efflux from the cell. Adenosine will terminate supraventricular tachyarrhythmia, which requires the AV node as a part of its re-entrant circuit. In cases in which rapid ventricular rates obscure atrial activity on the ECG, adenosine can induce transient AV nodal blockade and reveal the underlying atrial waveforms, resulting in establishment of the etiology of the arrhythmia. In this case, typical flutter waves are seen, negative-positive in the inferior leads and positive in lead V1. The flutter rate is 240 bpm, which is slightly slower than typical atrial flutter. This is likely the result of flecainide, which

continues

is a class IC agent that slows conduction, resulting in a slower flutter rate. Hence the arrhythmia is characteristic of typical or type I atrial flutter.

Although the patient was receiving flecainide as therapy for atrial fibrillation, it is not uncommon for a recurrence of arrhythmia to present with atrial flutter during therapy with an anti-arrhythmic agent. These drugs “stabilize” the atrial myocardium and eliminate the ability to sustain multiple reentrant circuits, as is seen with atrial fibrillation. In addition, it is possible that this patient has had atrial flutter in

the past that resulted in atrial fibrillation. The use of anti-arrhythmic agents often prevents atrial flutter from precipitating atrial fibrillation.

The atrial rate in atrial flutter is generally 260 to 320 bpm, and at this rate the normal AV node will not be able to conduct in a 1:1 relationship. Physiologic heart block will result in an incremental ratio of conduction (2:1, 3:1, 4:1, etc), with the ventricular rate being some fraction of the atrial rate ($\frac{1}{2}$, $\frac{1}{3}$, $\frac{1}{4}$, etc). Class I agents, particularly the class IC agents flecainide and propafenone, markedly slow conduction by reducing the upstroke of phase 0 of the fast action potential. As a result, there is a slowing of reentrant conduction velocity, which can

slow the atrial flutter rate. When the atrial flutter rate is decreased, there is less concealed conduction within the AV node and, therefore, there is a greater potential for 1:1 conduction. That appears to have been the case with this patient and accounts for the pulse rate of 240 bpm that was noted by the emergency department physician when the patient first presented.

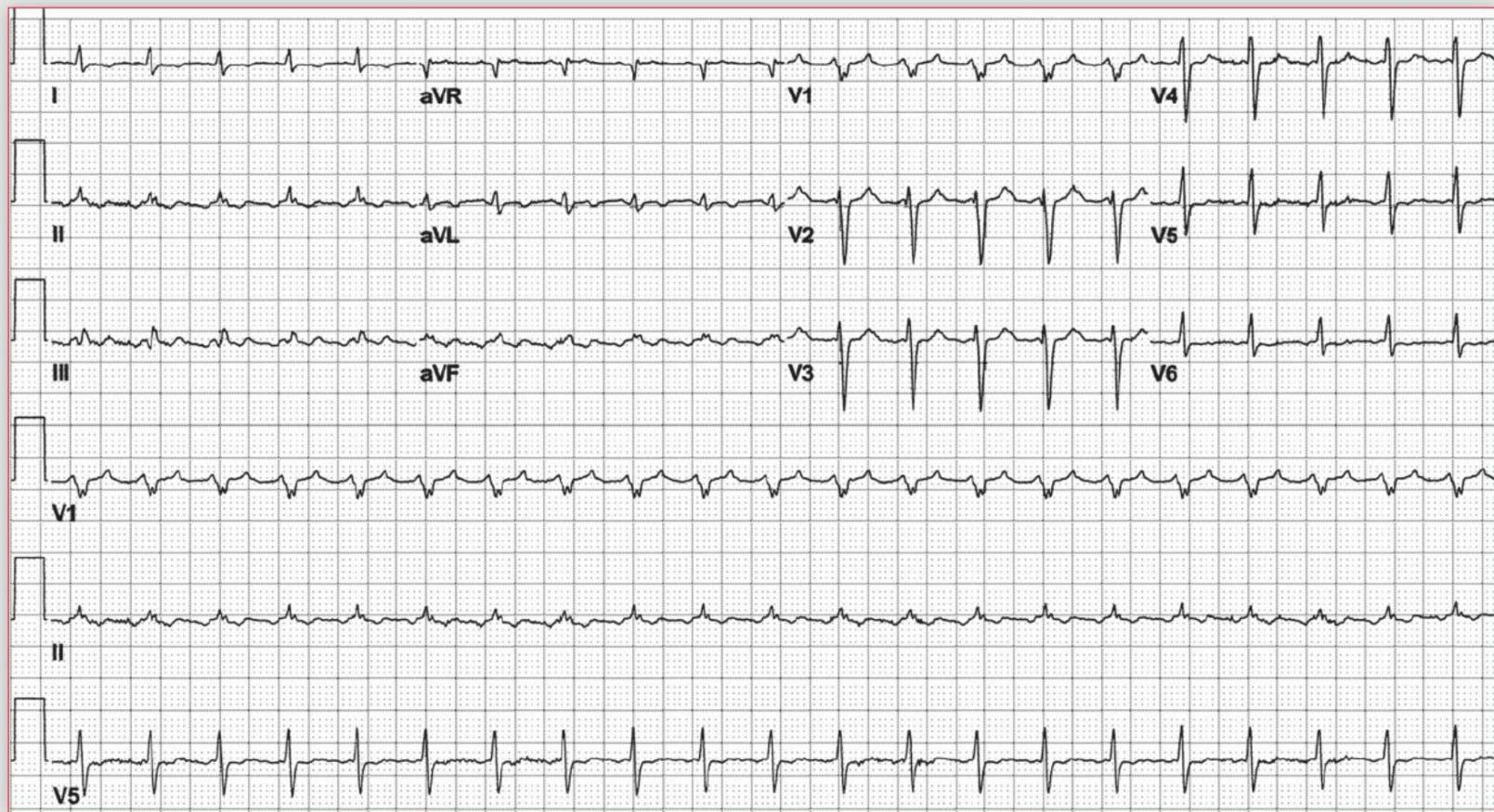
The half-life of adenosine is approximately 6 seconds, and so the marked heart block seen on this ECG will be transient; 1:1 AV conduction is likely to recur. A β -blocker or calcium-channel blocker should

be administered for acute control of the rapid ventricular rate. The patient should be placed on long-term β -blocker therapy in addition to flecainide to prevent the likelihood of 1:1 AV conduction when he is in atrial flutter. The choice of flecainide should be reconsidered as now there is also a diagnosis of atrial flutter. Options include an increase in the dose of flecainide, the use of another anti-arrhythmic agent, or consideration of atrial flutter ablation. ■

Core Case 24

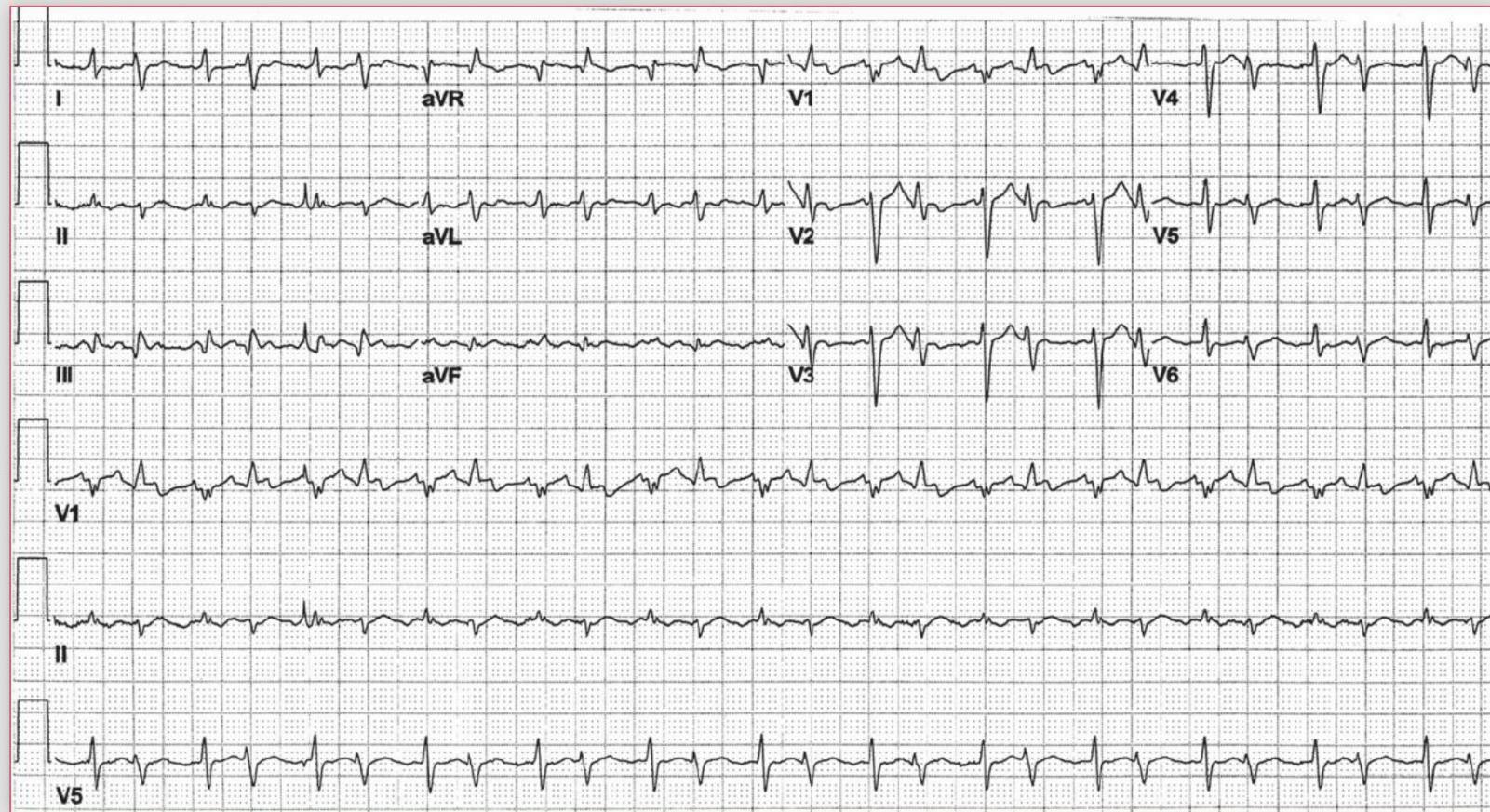
A 21-year-old woman with familial dilated cardiomyopathy presents to the local emergency department after she noted becoming inordinately breathless while performing Pilates exercises. She states that over the past few nights she has noted some breathlessness when lying down to sleep. On exam, she appears comfortable sitting upright. Her vital signs

ECG 24A



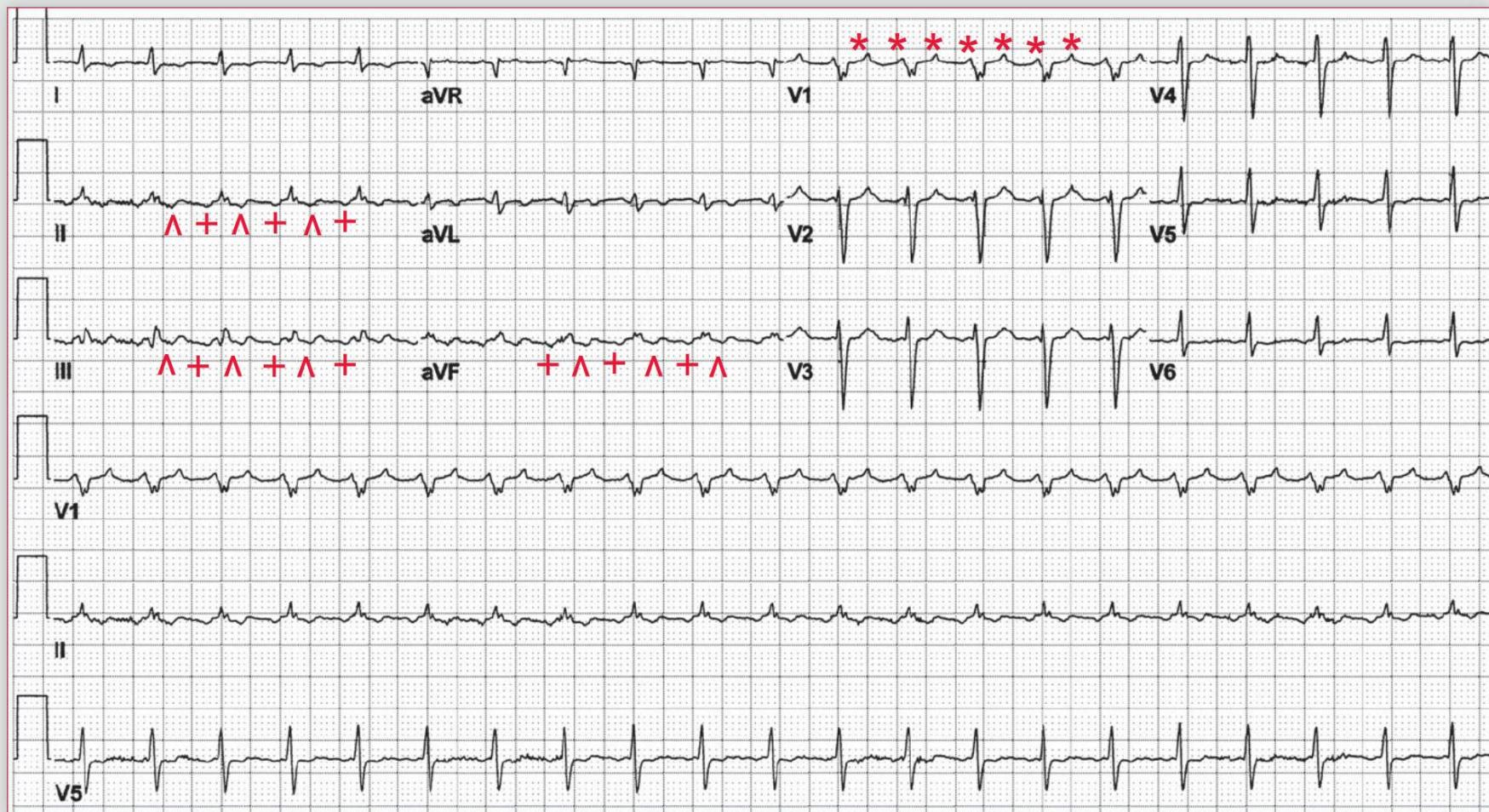
are notable for a heart rate of 140 bpm. Her cardiopulmonary exam is notable for a jugular venous pressure of 14 mm Hg with a monophasic waveform, bibasilar rales, and an S3 gallop. The rest of her exam is unremarkable. An ECG is obtained on presentation (ECG 24A), and then again when the nurse notices a change in the rate and QRS complex waveform on telemetry (ECG 24B).

ECG 24B



What are the findings on her ECGs?

What explains the change in waveform the nurse noticed?



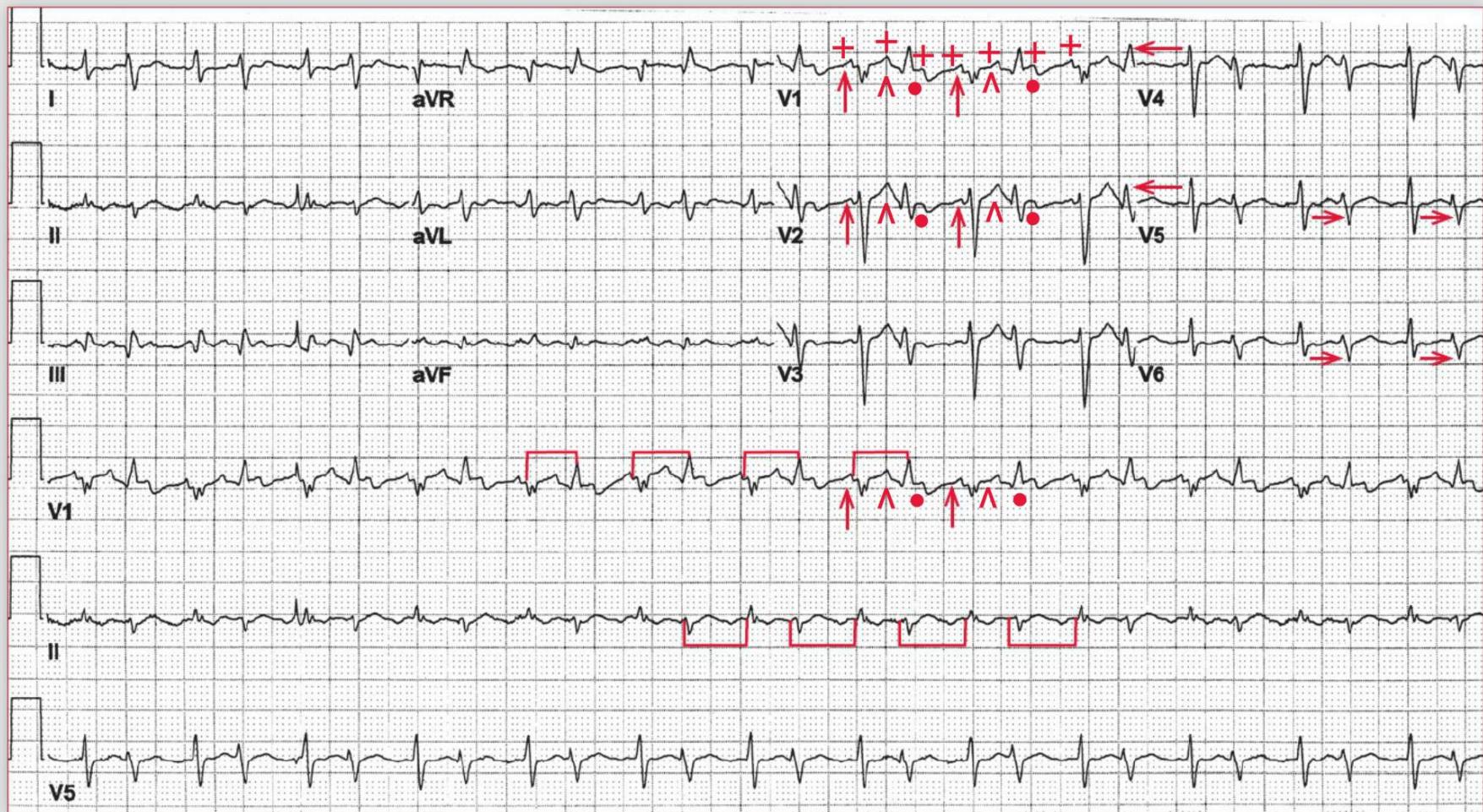
ECG 24A Analysis: Atrial flutter with 2:1 conduction, low-voltage limb leads

ECG 24A shows a regular rhythm at a rate of 140 bpm. The QRS complexes are normal in duration (0.10 sec) and have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (300/460 msec and 280/430 msec when the slightly prolonged QRS complex duration is considered). The limb leads have low voltage (*ie*, QRS complex < 5 mm in amplitude in each limb lead).

There is evidence of atrial activity (+), primarily seen in leads II, III, and aVF, with a negative waveform just before each QRS complex.

A second atrial waveform (Λ) can be seen within the ST segment, immediately after each QRS complex, resembling a T wave. However, this waveform has the same morphology as the waveform before the QRS complex and occurs at a regular interval. Hence the atrial rate is regular at 280 bpm and, therefore, this is atrial flutter with 2:1 AV conduction. In addition, two flutter waves (*) can be seen in lead V1; one is just before the QRS complex, resulting in a slurring of the upstroke of the QRS complex and resembling a broad R wave, and the second follows the QRS complex and could be confused with the T wave.

continues



ECG 24B Analysis: Atrial flutter with 3:2 Wenckebach (Mobitz type I second-degree AV block), rate-related right bundle branch block, low-voltage limb leads

In ECG 24B, there is a regularly irregular rhythm with a repeating pattern of long (◻) and short (▨) RR intervals; there is an appearance of group beating. Evidence of atrial activity can be seen, especially in lead V1 (+). The atrial rate is regular at 280 bpm. The rate and morphology of the atrial waveforms are the same as those in ECG 24A. Therefore, this is atrial flutter and there is variable AV conduction.

After the longer RR interval, the interval between the flutter wave and QRS complex (PR interval) is short (↑). The interval then lengthens before the next QRS complex (Λ), and then the third flutter wave is non-conducted (●). This is a pattern of 3:2 Wenckebach, which then repeats itself, giving the appearance of group beating. The QRS complex after the longer interval has the same morphology as was seen in ECG 24A, while the other QRS complex after the shorter RR interval is different; it has a tall R wave in leads V1 and V2 (←) and a broad S wave

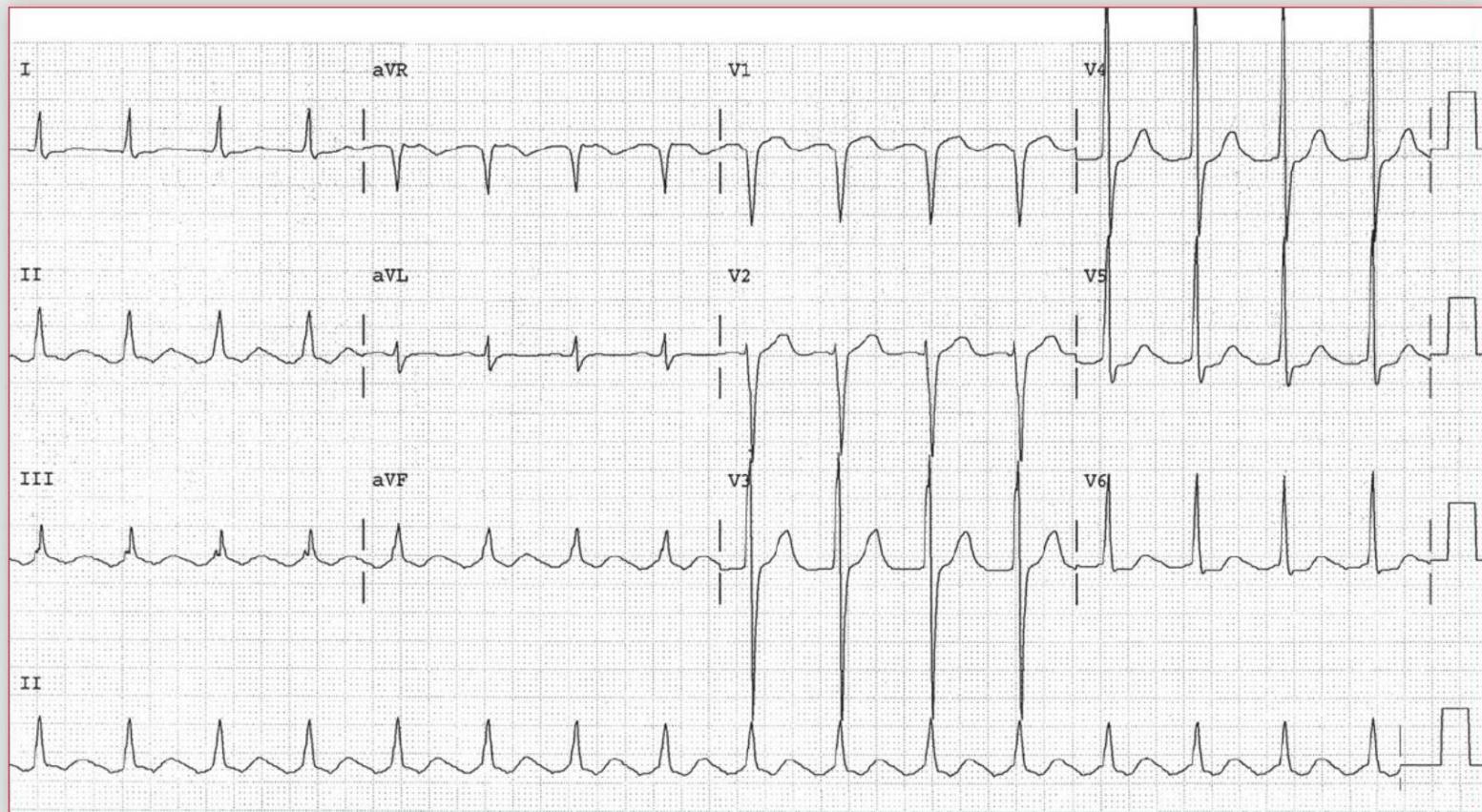
in leads I and V5-V6 (→). In addition, this QRS complex duration is slightly longer (0.12 sec) and has a right bundle branch block pattern, which is, therefore, rate related. Hence this ECG shows atrial flutter with 3:2 Wenckebach and a rate-related right bundle branch block.

Wenckebach is the result of decremental conduction through the AV node; that is, at faster rates there is progressive reduction in the rate of impulse conduction through the node, accounting for the progressive increase in the PR interval that is characteristic of Wenckebach. Although Wenckebach is most commonly observed during sinus rhythm, it may be seen with atrial arrhythmias, including atrial tachycardia or atrial flutter, in which there is rapid AV nodal activation. With these arrhythmias, impulse conduction to the ventricles is dependent on transmission through the AV node, similar to the situation with sinus rhythm. ■

Core Case 25

A 92-year-old woman presents to your clinic with a complaint of fatigue for the past several days. Upon further questioning, she notes exertional dyspnea, which she has never experienced before. She has a history of longstanding hypertension and asymptomatic Mobitz type II second-degree AV block for which

ECG 25A

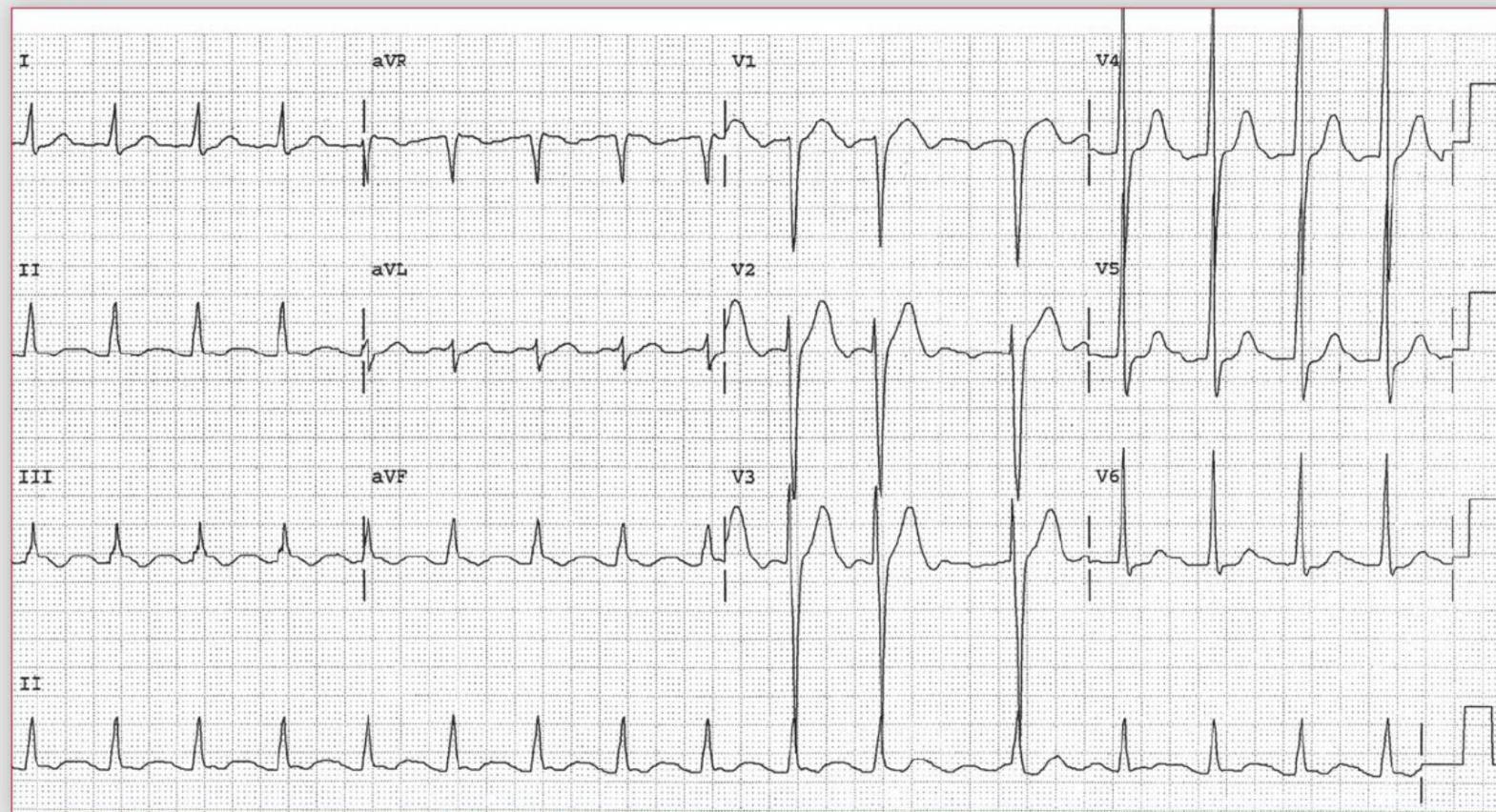


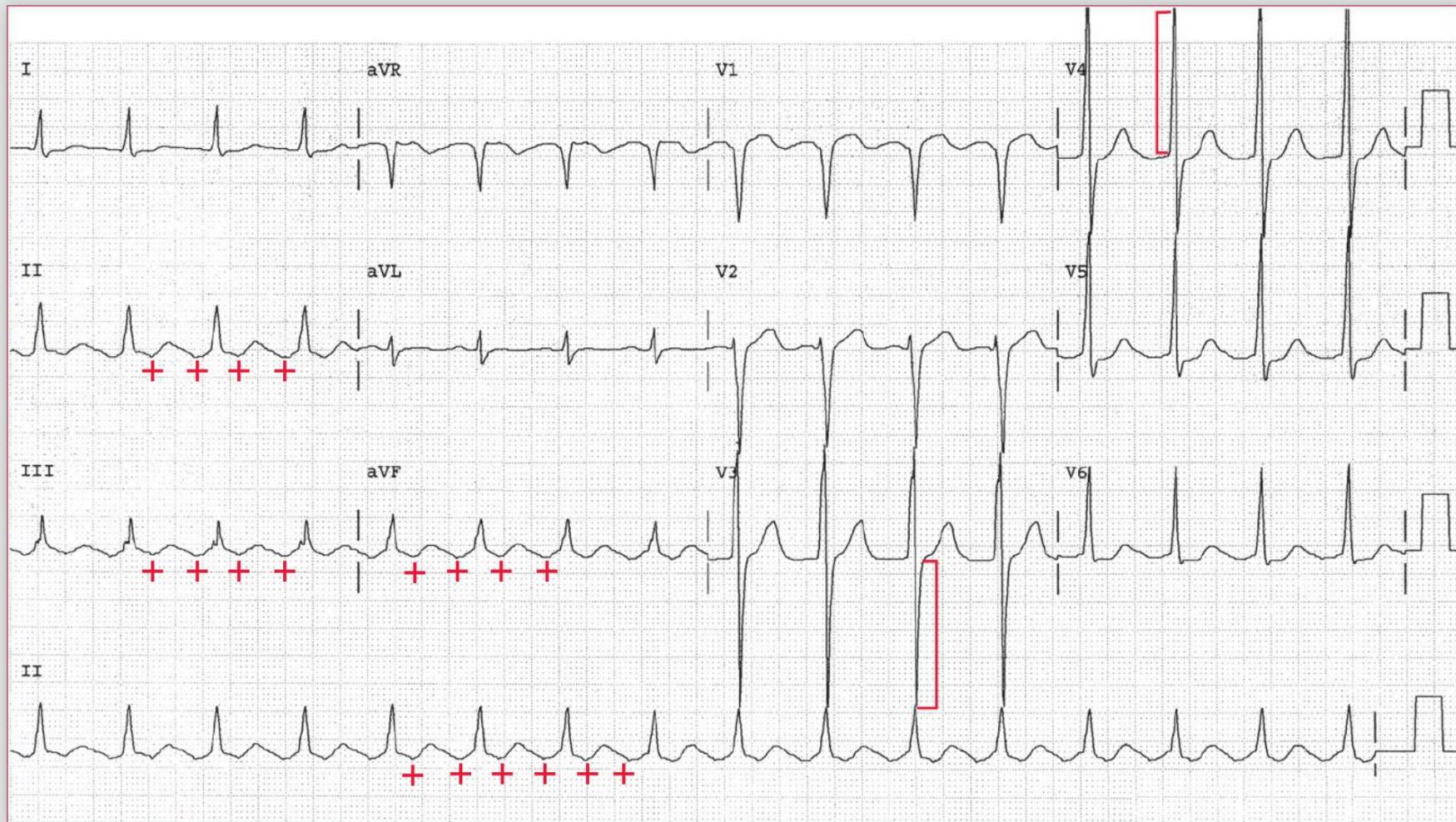
she received a pacemaker some years ago. On exam, her heart rate is regular at 100 bpm and her blood pressure is normal. Her cardio-pulmonary exam is also normal. Your medical technician hands you an ECG (25A) and states that he thinks it shows sinus tachycardia. The technician then hands you a second ECG (25B).

Do you agree that ECG 25A shows sinus tachycardia? If not, what is the reason for the elevated heart rate?

Does ECG 25B help you make a diagnosis?

ECG 25B



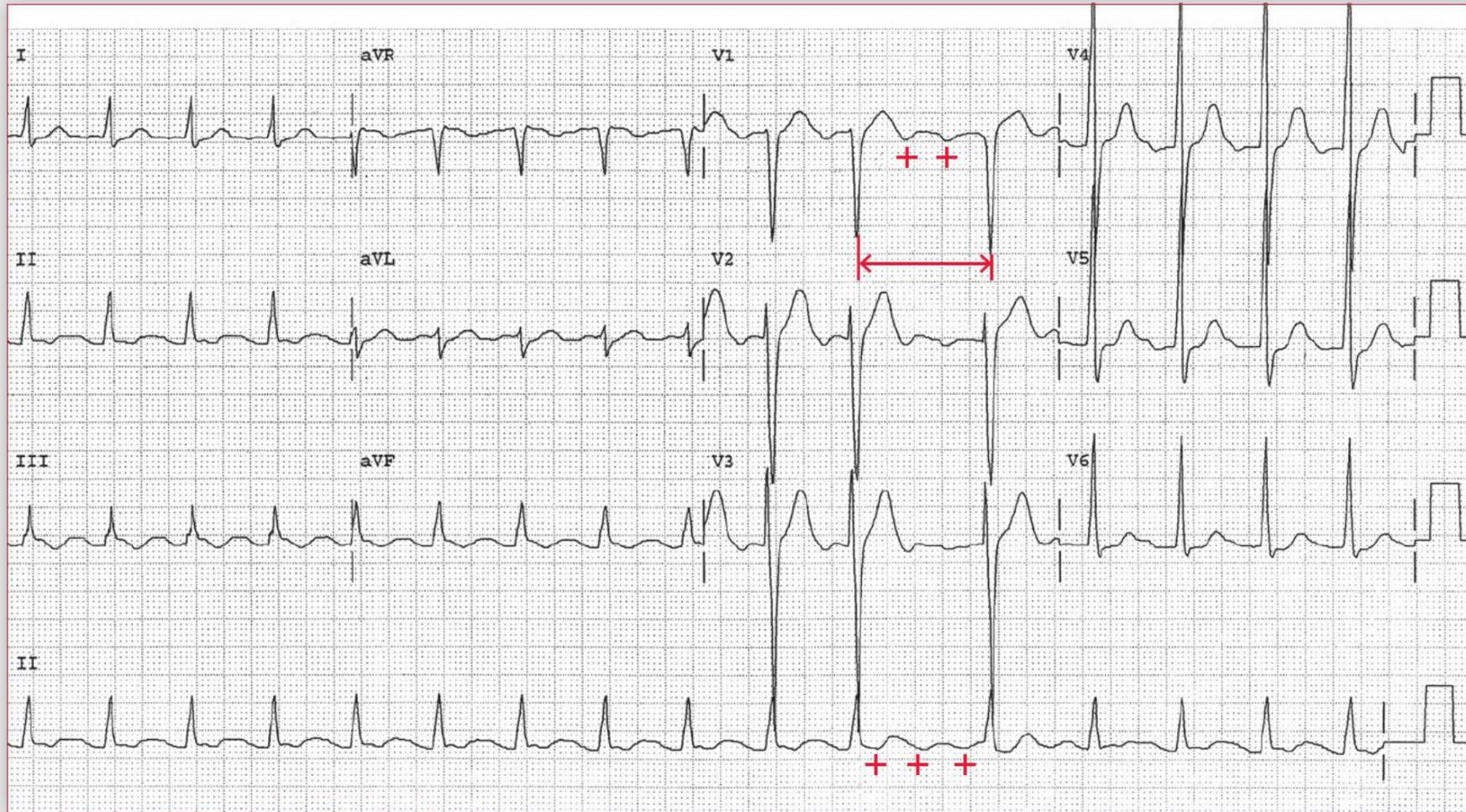


ECG 25A Analysis: Clockwise type I (typical) atrial flutter, left ventricular hypertrophy (LVH)

ECG 25A shows a regular rhythm at a rate of 100 bpm. The QRS complex duration is normal (0.10 sec) and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS amplitude is increased (S-wave depth in lead V3 = 28 mm [] and R-wave amplitude in lead V4 [] = 30 mm), which is consistent with left ventricular hypertrophy (*ie*, S-wave depth in lead V3 + R-wave amplitude in

lead V4 = 58 mm). The QT/QTc intervals are normal (350/460 msec). Although no clear P waves are seen, there are prominent undulations between each QRS complex in leads II, III, and aVF (+). They are occurring at regular intervals at a rate of 200 bpm. These waveforms are suggestive of atrial flutter, even though the rate is relatively slow for flutter.

continues



ECG 25B Analysis: Clockwise type I (typical) atrial flutter, slow atrial flutter rate, LVH

In ECG 25B the rhythm is basically regular at a rate of 100 bpm, although there is one long RR interval (↔). During this long interval three sequential atrial waveforms can be seen (+); these are at regular intervals at a rate of 200 bpm, identical to the rate of the waveforms noted in ECG 25A. These waveforms show continuous undulation without any isoelectric baseline between them, and they are atrial flutter waves.

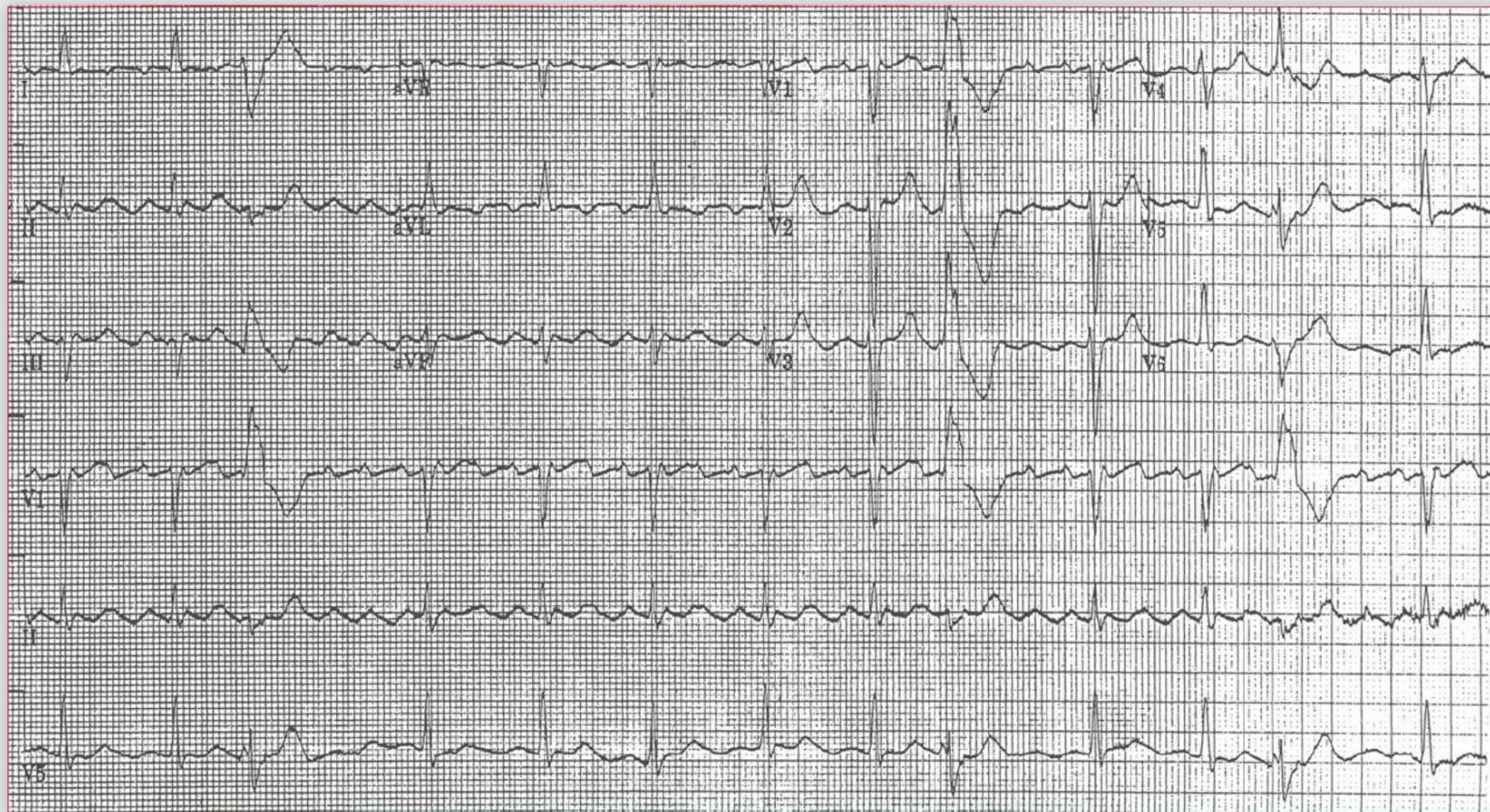
The diagnosis of flutter is established primarily by the atrial rate. Atrial flutter occurs at a rate faster than 260 bpm and is the only atrial arrhythmia with a rate this rapid. However, the rate of atrial flutter

may be less than 260 bpm as a result of anti-arrhythmic drug therapy or disease of the atrial myocardium. Even if the rate is slower than 260 bpm, the typical flutter morphology is maintained (*ie*, continuous undulations without any isoelectric baseline between the waveforms). In contrast, atrial tachycardia has distinct P waves with an isoelectric baseline between the P waves. Therefore, although the atrial rate is only 200 bpm in this case, the waveforms are typical of atrial flutter. It is possible that in this patient the longstanding hypertension and the presence of left ventricular hypertrophy have caused hypertrophy and fibrosis of the atrial myocardium, resulting in the slower flutter rate. ■

Core Case 26

A 72-year-old man is admitted to the electrophysiology suite for direct current cardioversion of a supraventricular arrhythmia that is causing him significant symptoms. ECGs before (26A) and after (26B) cardioversion are presented.

ECG 26A

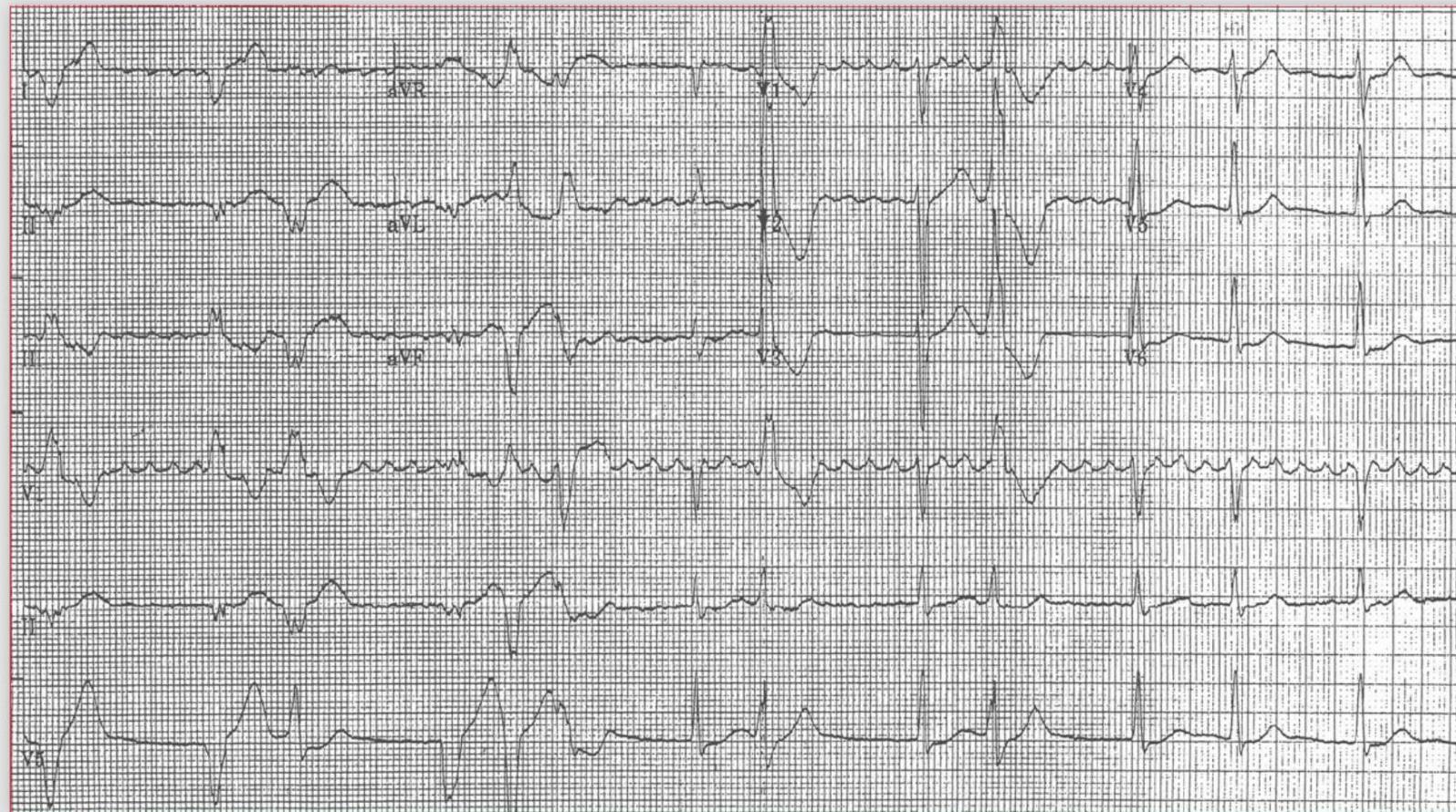


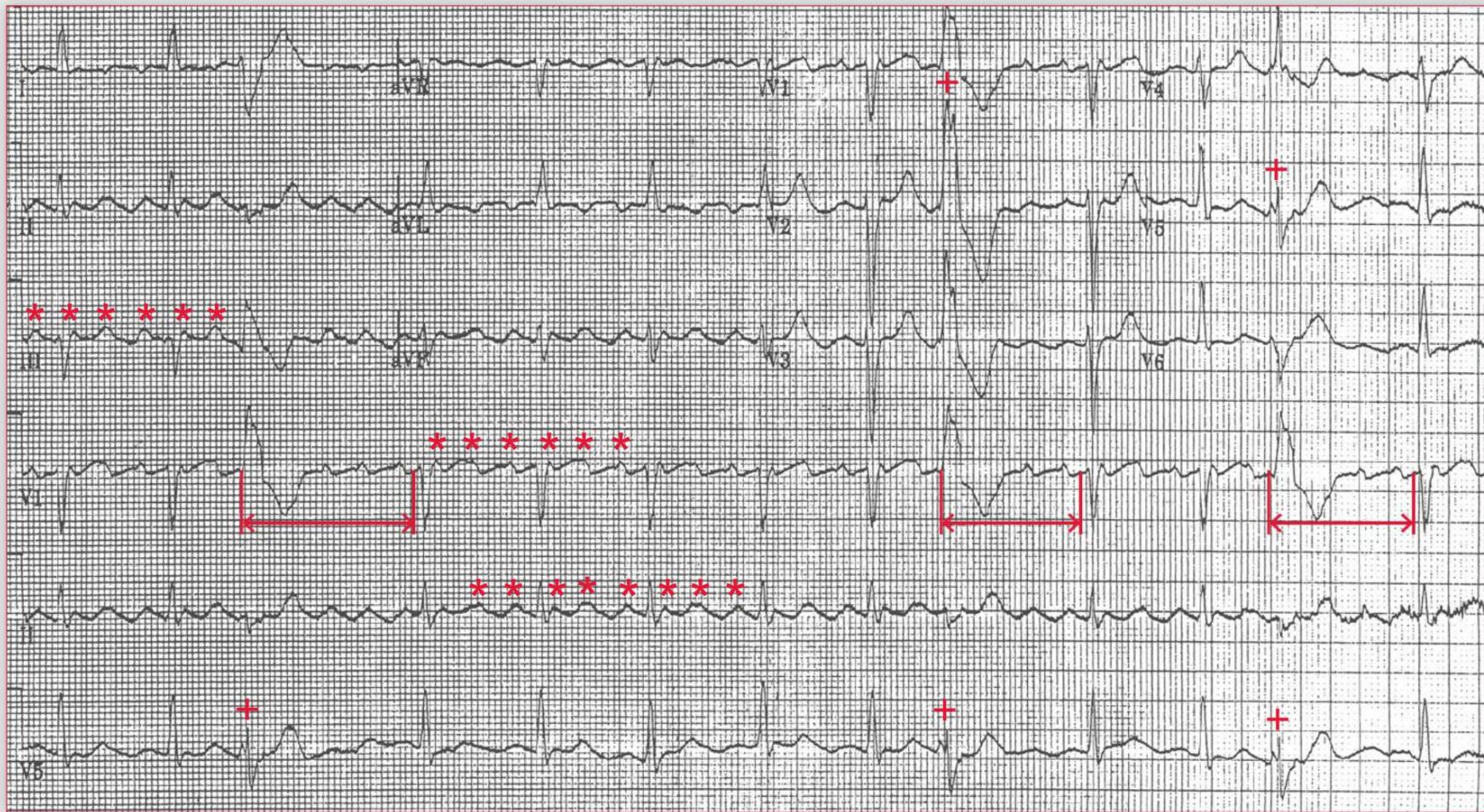
What is his bothersome arrhythmia?

What is the outcome of the cardioversion?

What is the next step in this patient's therapy?

ECG 26B





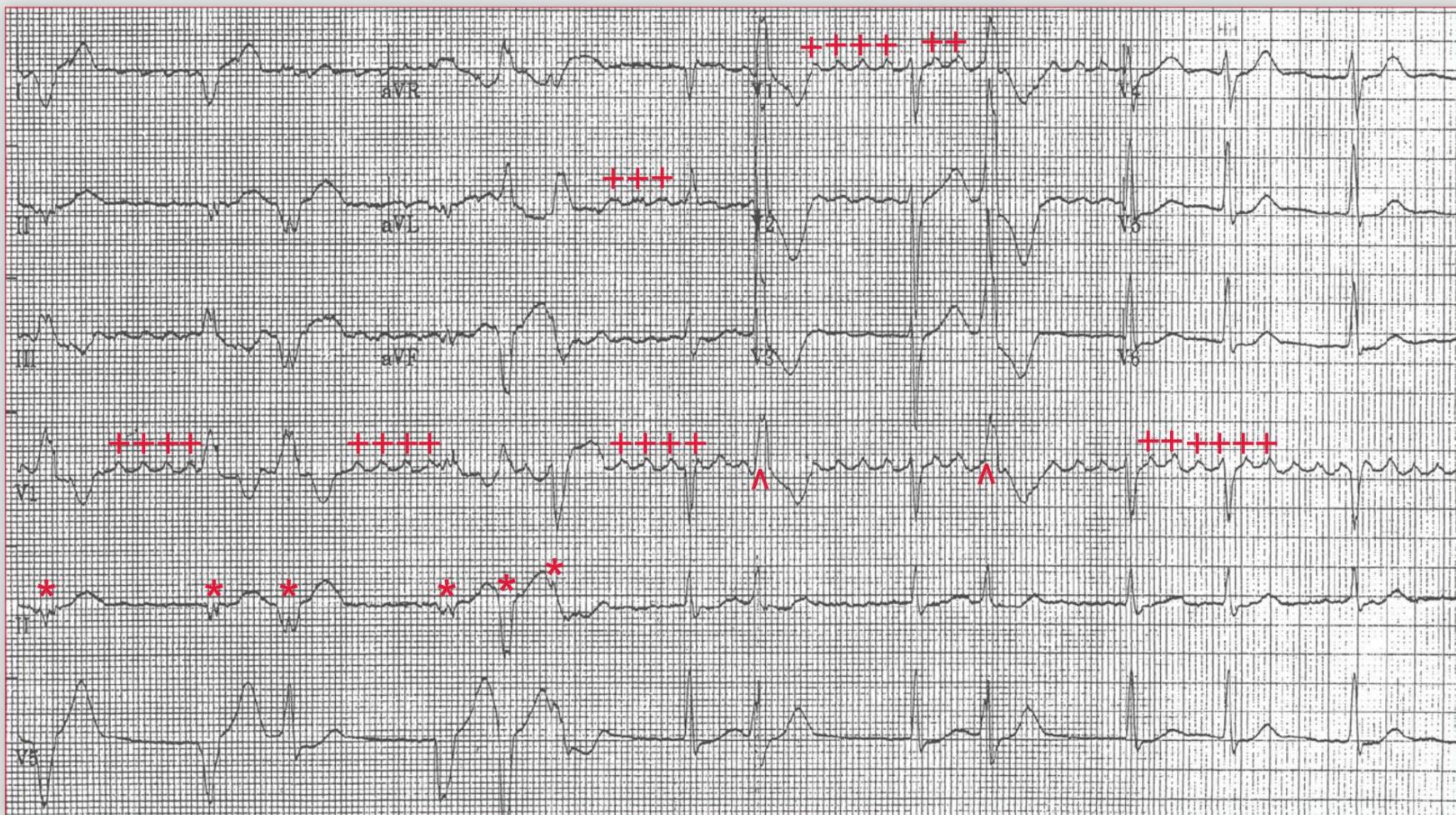
ECG 26A Analysis: Clockwise type I (typical) atrial flutter,
premature ventricular complexes, left anterior fascicular block

In ECG 26A the rhythm is basically regular at a rate of 75 bpm. However, there are three short RR intervals as a result of premature complexes (+) that are wide and abnormal. These are premature ventricular complexes. After the premature complexes there is a long RR interval (↔), representing what would be termed a compensatory pause (*ie*, the RR interval around the premature ventricular complex is the same as two RR intervals). The QRS complex morphology and duration (0.08 sec) are normal. The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative

QRS complex in leads II and aVF with an rS morphology). This is a left anterior fascicular block. The QT/QTc intervals are normal (380/420 msec).

Seen between each QRS complex are atrial waveforms that are continuously undulating and “saw tooth” (*). These waveforms are completely regular and uniform in morphology at a rate of 300 bpm. They are negative-positive in leads II, III, and aVF and hence represent typical atrial flutter with 4:1 AV conduction.

continues



ECG 26B Analysis: Type II (atypical) atrial flutter, ventricular ectopy

ECG 26B, obtained after cardioversion, shows a change in the atrial waveforms. The atrial waveforms are upright (+) rather than negative-positive in leads II, III, aVF, and V1. In addition, the atrial rate has increased to approximately 340 bpm. The rhythm is still atrial flutter, but it is now atypical flutter.

As with typical atrial flutter, the atrial waveforms of atypical atrial flutter are uniform in morphology, amplitude, and interval. There is no isoelectric baseline between the atrial flutter waves, and they are continuously undulating (saw tooth). However, the atrial rate is higher than 320 bpm and the flutter waves are positive in leads II, III, and aVF. The first six QRS complexes (*) are wide and bizarre and are ventricular complexes. Thereafter, there are two additional premature ventricular complexes (Δ).

In general, low energy is effective for reverting typical atrial flutter. Often an energy level of only 25 joules (biphasic cardioverter) is effective. However, typical flutter can convert to atypical flutter if insufficient energy is used, although the exact mechanism for this is unclear. Atypical flutter is more difficult to revert as the circuit results from a small area of slow conduction due to a functional change in membrane refractoriness. In this situation, the circuit is small and conducts more rapidly, and there is only a very small excitable gap (*ie*, small area of the myocardium in which there is slow conduction). Cardioversion or overdrive pacing is less likely to be effective as there is less time for an impulse to enter the circuit and depolarize the myocardium, thereby interfering with the circuit and terminating the arrhythmia. Higher energies are often necessary to depolarize the entire atrial myocardium and hence allow for sinus rhythm to be restored. ■

Notes

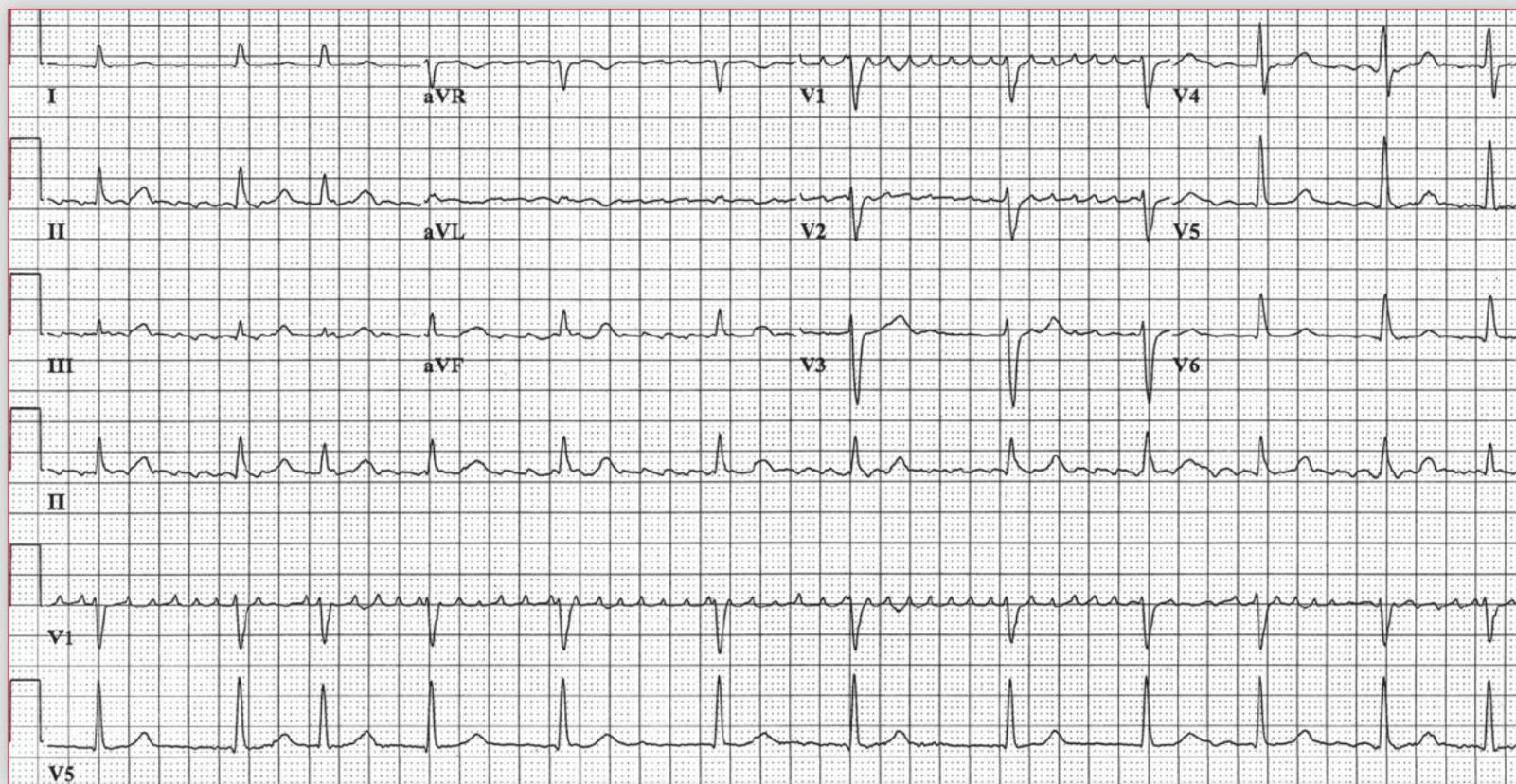
A 78-year-old man with a history of diabetes and silent coronary disease presents to the hospital with progressive angina. Coronary angiography reveals multi-vessel coronary disease, and he undergoes coronary artery bypass grafting. His surgery is uncomplicated, and he is extubated and off vasopressors and inotropes within the first 24 hours after the procedure. On postoperative day 3, however, he complains of feeling “unwell” and admits to some dyspnea. The primary surgical team caring for him obtains an ECG.

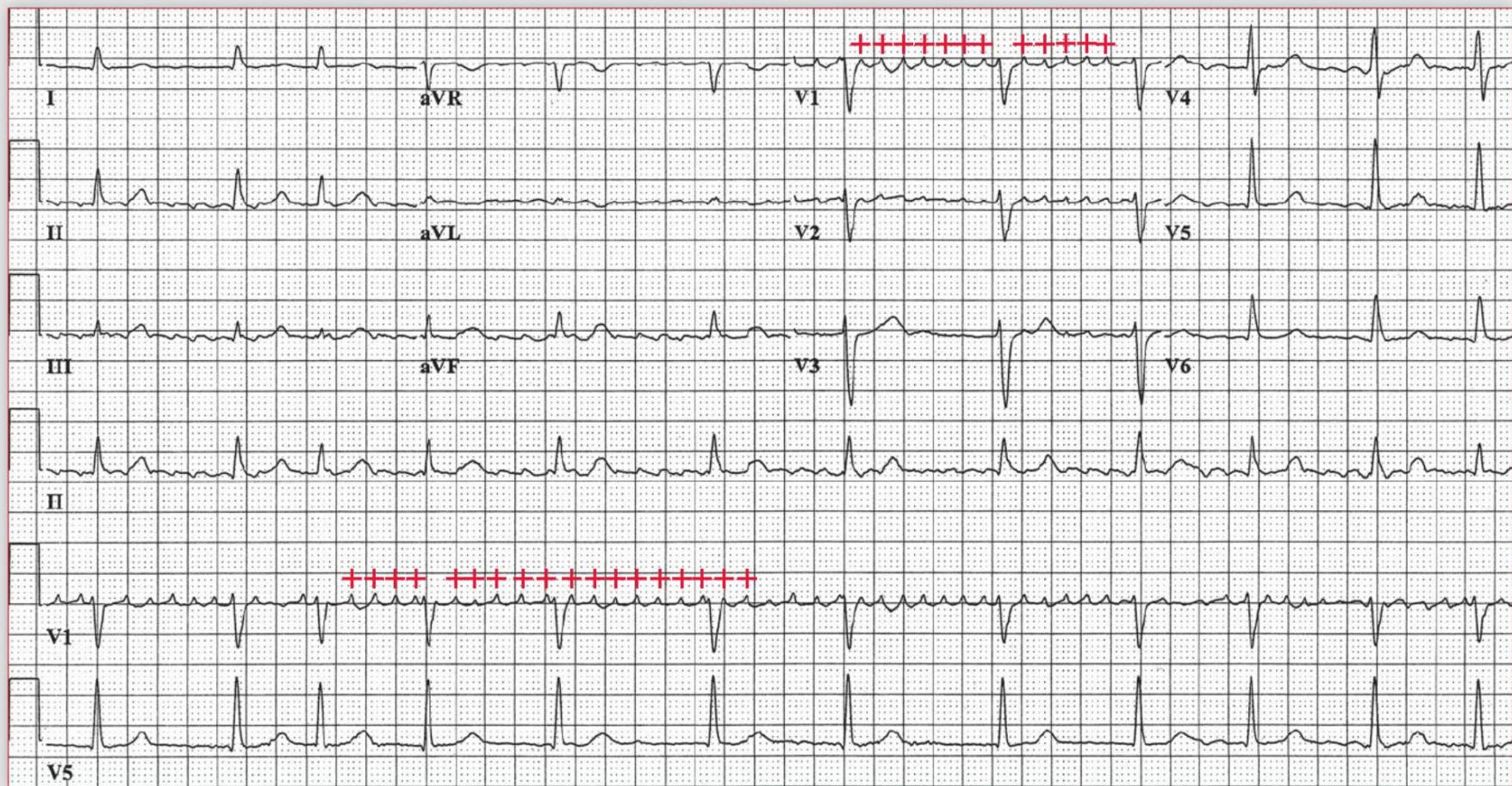
What are the findings on this ECG?

What are the implications, if any, for the patient's overall prognosis?

What therapy, if any, is indicated?

Can anything have been done to prevent this occurrence?





ECG 27 Analysis: Coarse atrial fibrillation with moderate ventricular response, intraventricular conduction delay

The rhythm is irregularly irregular, and the average rate is 72 bpm. The QRS complexes are widened at 0.12 second with a normal morphology. Hence there is an intraventricular conduction delay. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (440/480 msec) but are normal when corrected for the prolonged QRS complex duration (400/440 msec).

No organized atrial activity is seen, but there are rapid atrial waveforms that are irregular in morphology, amplitude, and interval (+). Although these waveforms resemble those seen in atrial flutter, the waveforms in atrial flutter are uniform in morphology, amplitude, and interval, unlike what is seen on this ECG. In addition, the rhythm is irregularly irregular, unlike what is seen with atrial flutter, which is regular or regularly irregular (*ie*, there is a pattern to the RR intervals). Hence the underlying atrial rhythm is coarse atrial fibrillation and not

atrial flutter. In general, coarse fibrillatory waves are associated with atrial fibrillation that has a more recent onset (*ie*, weeks or months), while they become finer when atrial fibrillation is of longer duration (*ie*, months or years).

Atrial fibrillation after coronary artery bypass graft (CABG) surgery is common, generally occurring between postoperative days 2 and 3. The incidence varies between 20% and 40% and increases to 60% when valve surgery is also performed. It has been reported that the presence of postoperative atrial fibrillation may be associated with an increased mortality rate 30 days postoperatively and at 4- to 5-year follow-up. Although an association with increased mortality is not certain, this arrhythmia is associated with increased morbidity and a longer hospital stay.

continues

A number of clinical risk factors have been reported to be associated with an increased risk for postoperative atrial fibrillation. Factors include a previous history of atrial fibrillation, increased left atrial size or cardiomegaly, long bypass and aortic cross-clamp times, previous cardiac surgery, chronic obstructive pulmonary disease, obesity, absence of β -blocker or angiotensin-converting enzyme inhibitor treatment or withdrawal of previous treatment, severe right coronary artery stenosis, or prolonged P-wave duration on the surface ECG (> 116 msec).

Surgery-related factors associated with postoperative atrial fibrillation include pericarditis, atrial injury from surgical handling or from cannulation, acute atrial enlargement from pressure or volume overload, inadequate cardioprotection during bypass, atrial infarction and/or ischemia, hyperadrenergic state, pulmonary complications, hypokalemia, and hypomagnesemia.

Therapies that may reduce the incidence of post-CABG atrial fibrillation include the use of preoperative β -blockers, statins, and amiodarone. Although the initiation of β -blockers and statins prior to

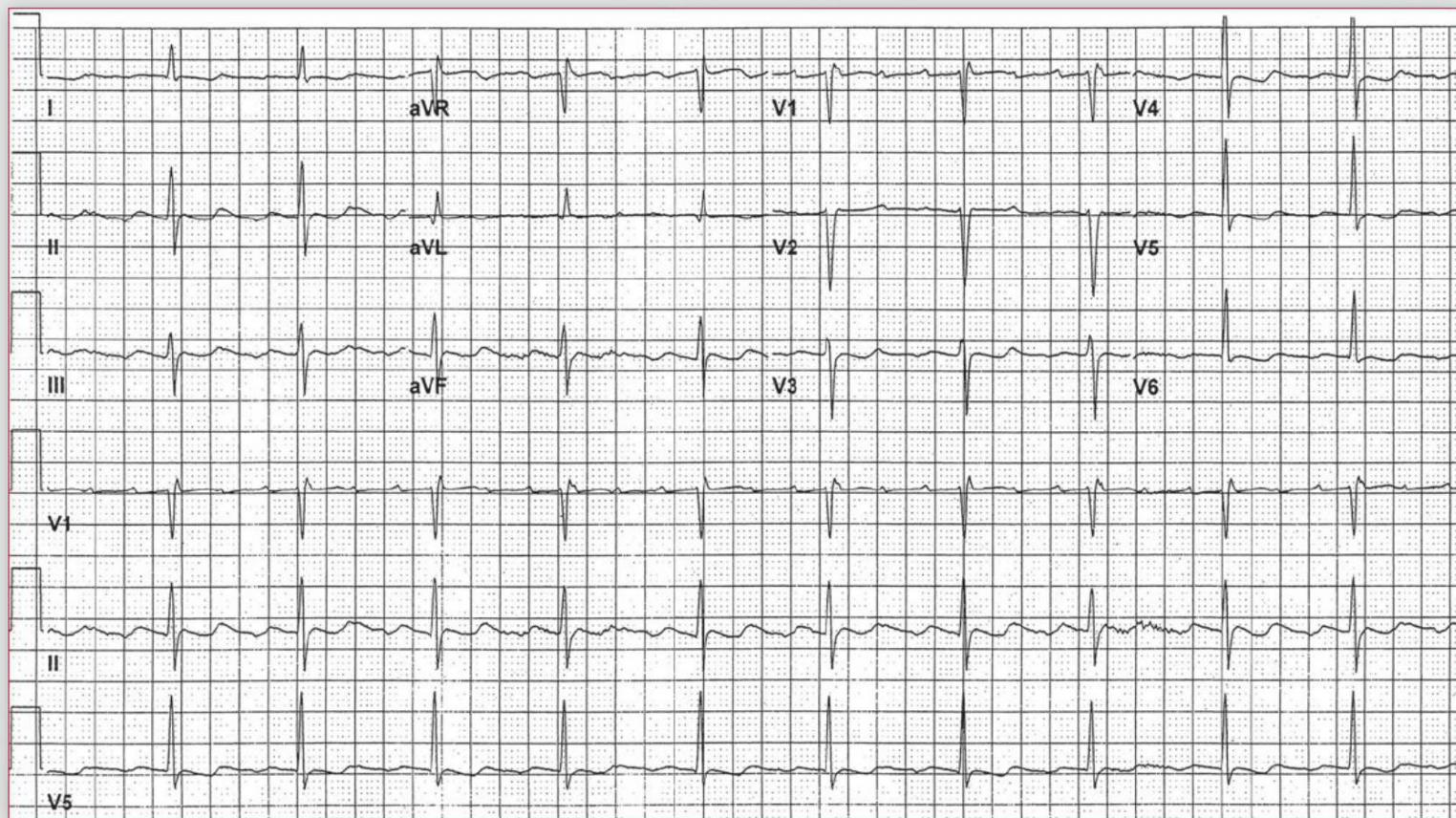
CABG surgery is routine, the institution of preoperative amiodarone therapy is more controversial and often not performed. Amiodarone must be started at least several days or longer before surgery to have any benefit. Amiodarone is usually reserved for the treatment of postoperative atrial fibrillation rather than for prophylaxis against this condition. Rate control is warranted; cardioversion is usually not required or necessary. As the cause of atrial fibrillation in these patients (provided they do not have preoperative atrial fibrillation) is often related to pericardial inflammation, myocardial injury, or

volume overload, patients tend to convert spontaneously to a normal sinus rhythm once these factors resolve. Anticoagulation should be initiated as prophylaxis against embolic stroke, although data for heparinization as a bridge to warfarin therapy are lacking. In cases of spontaneous conversion to sinus rhythm, the duration of anticoagulation is unknown but reasonable practice may include continuation of systemic anticoagulation for 4 weeks with discontinuation if outpatient Holter monitoring documents maintenance of a sinus rhythm. ■

Core Case 28

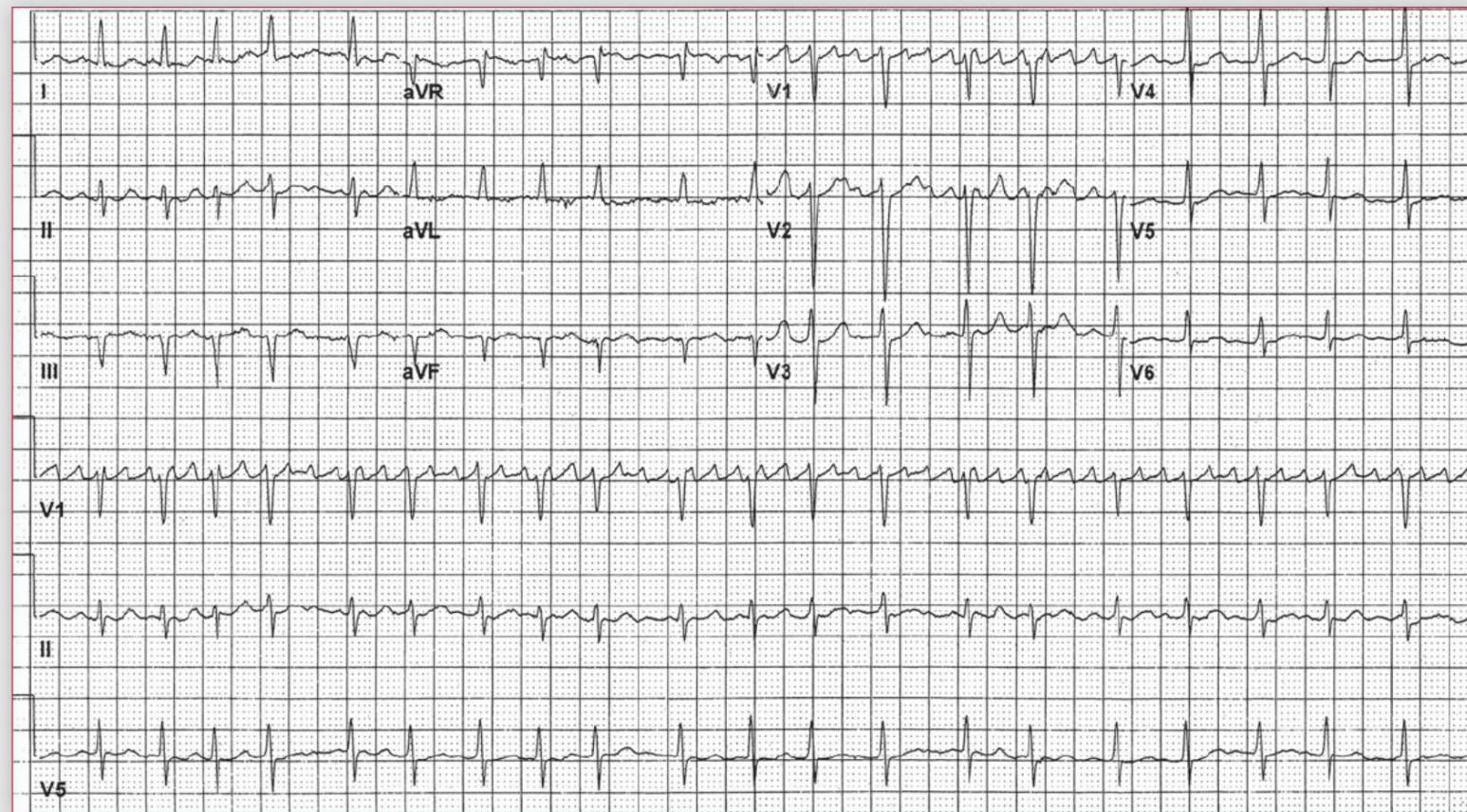
A 76-year-old man who underwent coronary artery bypass grafting and the cryomaze procedure for atrial fibrillation in the distant past is admitted with palpitations and exercise intolerance. He is usually able to run 3 to 5 miles most days of the week, but over the past few days has noted dyspnea that has limited his usual workouts. He has noted his pulse to be

ECG 28A



irregular at times as well. His exam is notable for a regular radial pulse. His physical exam is otherwise normal except for a well-healed midline surgical scar on the chest. The ECG technician has obtained a tracing for your review (ECG 28A). The following morning, the patient suddenly develops shortness of breath and palpitations. You obtain a second ECG (28B).

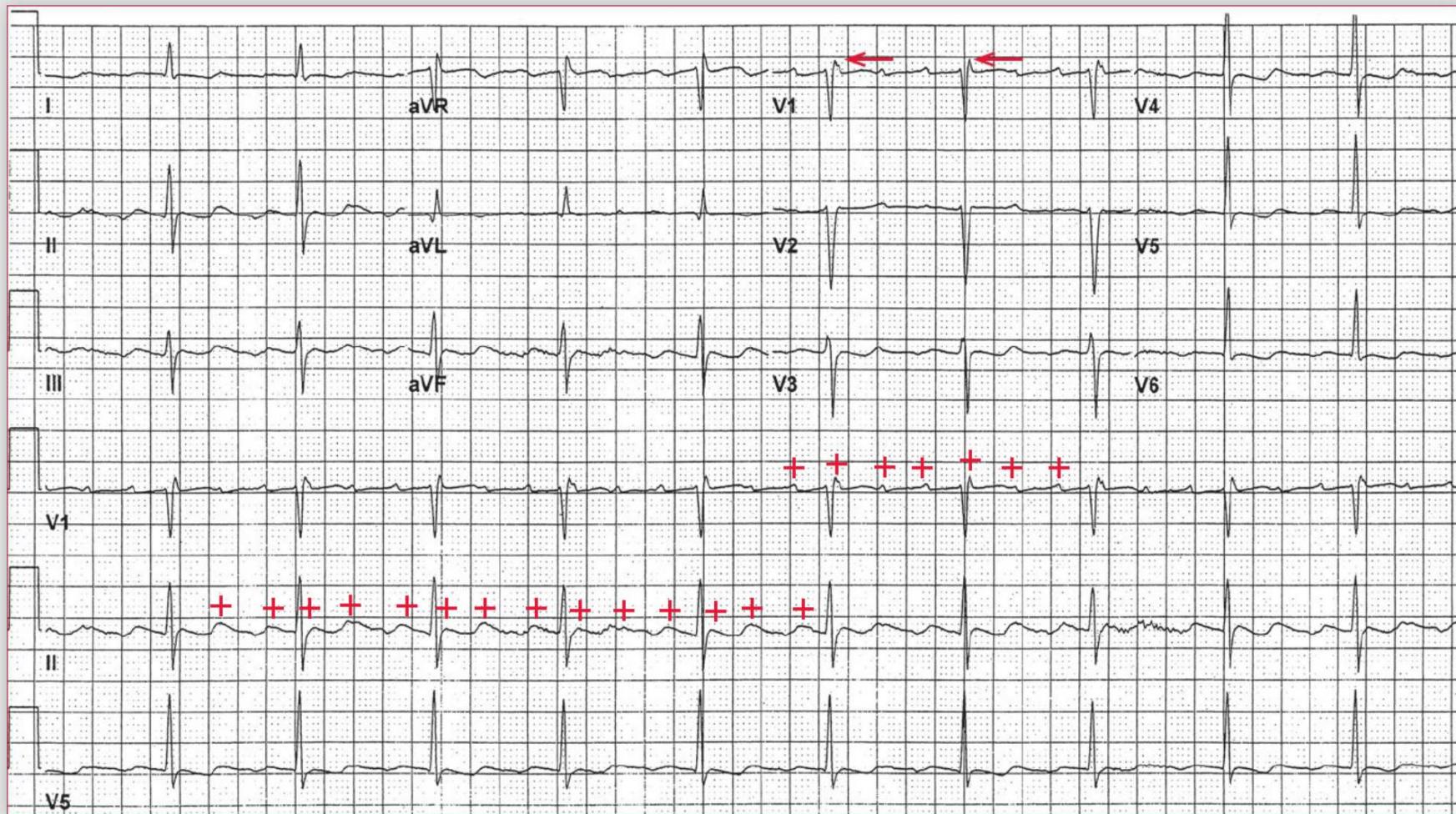
ECG 28B



What is the diagnosis?

What types of arrhythmia is the patient displaying?

Where are their anatomic origins?



ECG 28A Analysis: Slow atrial flutter at 220 bpm, left atrial in origin

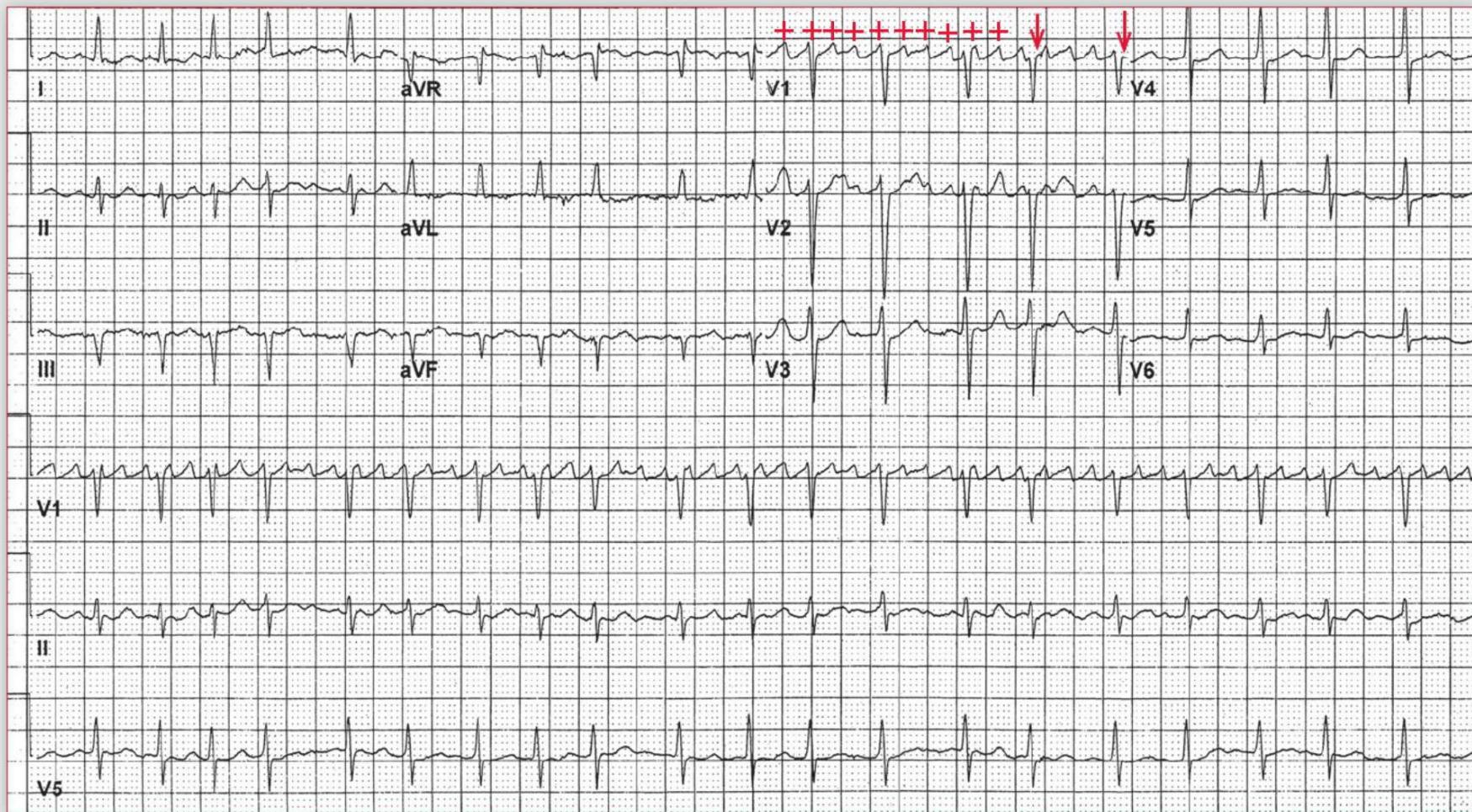
ECG 28A shows a regular rhythm at a rate of 68 bpm. Distinct atrial activity (+) can be seen at a rate of 220 bpm. The atrial waveforms are regular and of uniform morphology, amplitude, and interval; the waveforms are negative in leads II, III, and aVF. There is no isoelectric baseline between the waves (*ie*, the baseline is continuously undulating). Although there appear to be distinct P waves in lead V1, the baseline between them is not constant. Hence this is atrial flutter with a slow flutter rate and 3:1 AV conduction. Although not absolutely certain, it appears that the atrial waveforms are positive–negative in leads II and aVF. Hence this resembles atypical atrial flutter, although the rate is slow for this type of arrhythmia. This is more consistent with left atrial flutter, likely the result of previous left atrial surgery.

The QRS complex is narrow (0.08 sec) with a normal morphology and axis, about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). Noted in lead V1 is what appears to be a complex with an RSR' morphology (\leftarrow); however, by measuring the interval

between the flutter waves (or flutter rate), it can be determined that this is actually a superimposed flutter wave and not part of the QRS complex. The QT/QTc intervals are normal (420/450 msec).

The slow flutter rate may be due either to anti-arrhythmic drugs the patient is taking or to inherent disease of the atrial myocardium. Any process, organic or pharmacologic, that affects the activity of fast sodium channels within the myocardium and slow conduction will affect the flutter rate. It is also possible that this is left atrial flutter, as a result of a previous maze procedure in the left atrium. This is supported by the fact that the atrial morphology suggests an atypical flutter, although at a slow rate. Left atrial flutter may be micro- or macro-reentrant in etiology. When following left atrial ablation procedures (either surgical or catheter-based), the areas of scar within the left atrium create anatomic features that may allow for macro-reentrant circuits. This is the likely explanation for this patient's slow atrial flutter.

continues



ECG 28B Analysis: Coarse atrial fibrillation

In ECG 28B, obtained the following day, the rhythm is irregularly irregular and the ventricular rate averages 120 bpm. Prominent atrial waveforms are seen (+), primarily in lead V1. However, they are occurring at a more rapid rate (> 320 bpm) and are variable in morphology, amplitude, and interval. This is no longer atrial flutter, but is now atrial fibrillation with coarse fibrillatory waves. The QRS complex width,

axis, and morphology are identical to those seen in ECG 28A. The QT/QTc intervals are normal (280/400 msec). Also noted is that the QRS complex in lead V1 does not have an RSR' morphology; that is, there is no R' waveform present (↓). This is in contrast to what was seen in ECG 28A and confirms that this R' waveform is not part of the QRS complex but is indeed a flutter wave. ■

Notes

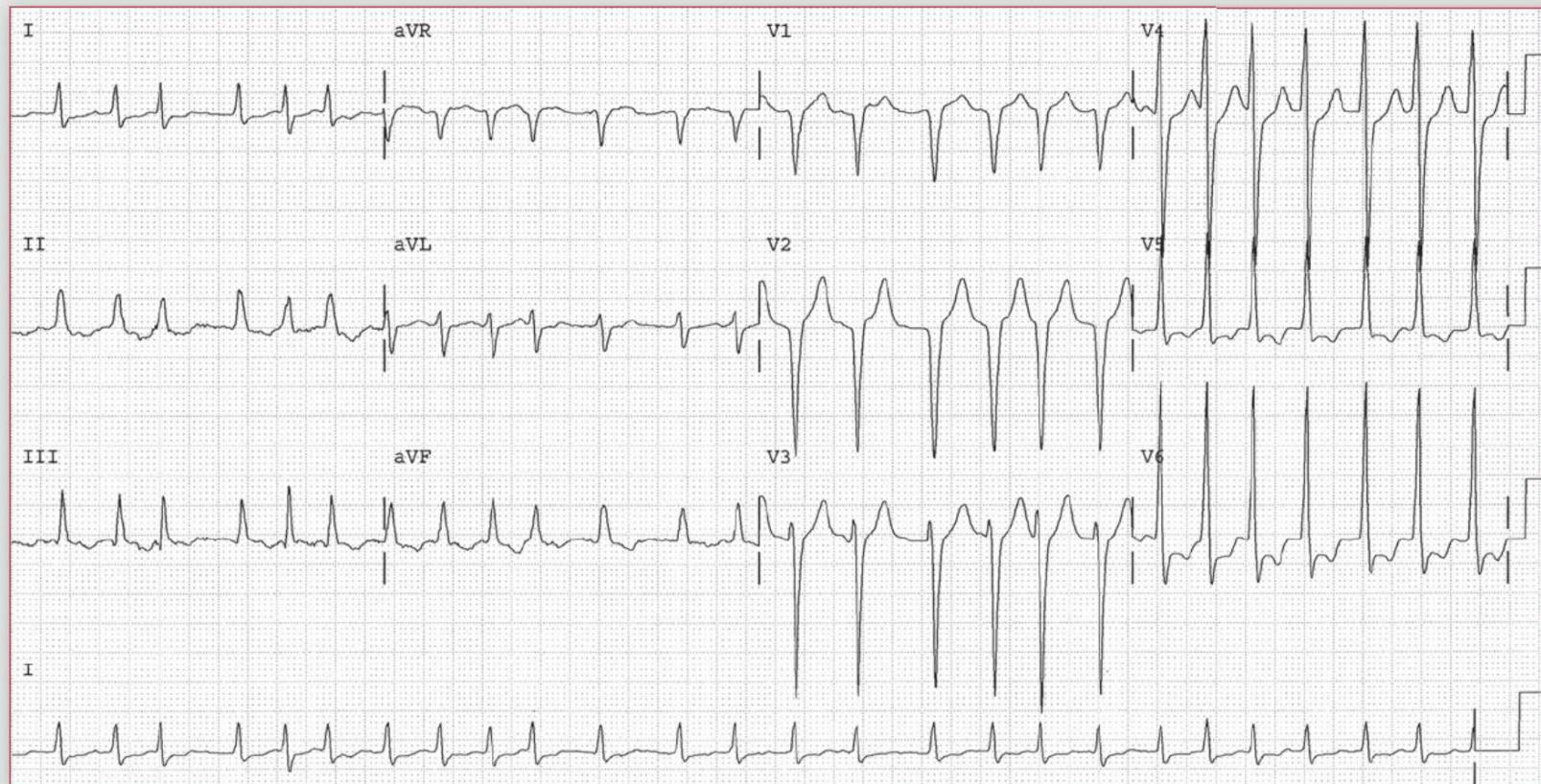
A 45-year-old man with obesity, hypertension, and obstructive sleep apnea presents to the emergency department with chest pain, dyspnea, and palpitations. His symptoms began suddenly this morning. He is a sedentary individual but denies exertional dyspnea or angina in the past.

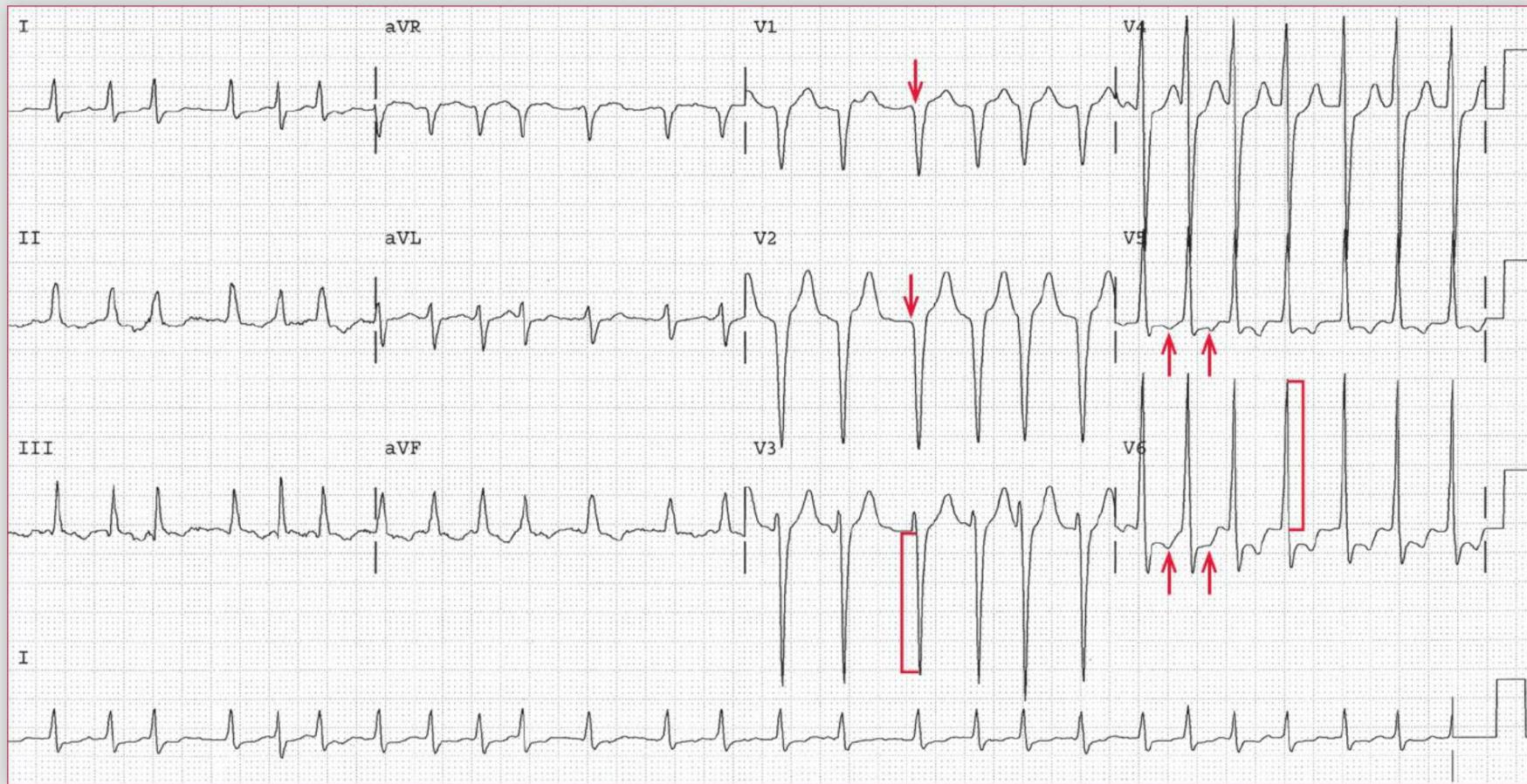
On exam, his heart rate is 160 bpm and his blood pressure is 100/60 mm Hg. His respiratory rate is 18 breaths/min, and his oxygen saturation level is 96% on room air. He appears to be in mild distress. His cardiopulmonary exam is normal except for a rapid and irregular radial pulse. You obtain an ECG.

What is the diagnosis?

What are the patient's risk factors for developing this arrhythmia?

What is the appropriate workup and the indicated short- and long-term therapy?





ECG 29 Analysis: Atrial fibrillation with rapid ventricular response, left ventricular hypertrophy, old anteroseptal myocardial infarction, ST-T wave abnormalities

The rhythm is irregularly irregular at an average rate of 156 bpm. There are only three rhythms that are irregularly irregular: sinus arrhythmia (one P-wave morphology and PR interval), multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) (three or more different P-wave morphologies without any P-wave morphology being dominant), or atrial fibrillation (no organized P waves). No obvious atrial activity is seen in any lead. Hence this is atrial fibrillation with fine fibrillatory waves.

The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex duration (0.10 sec) and morphology are normal. The amplitude of the QRS complex is increased (S-wave depth in lead V3 = 28 mm [] and R-wave amplitude in lead V6 = 25 mm []); S-wave depth in lead V3 + R-wave amplitude in lead V6 = 53 mm), which is diagnostic for left ventricular hypertrophy (LVH). In addition, there are no R waves in leads V1-V2 (↓), suggesting an anteroseptal myocardial infarction, although this may also be the result of LVH. The QT/QTc intervals are normal (280/450 msec).

ST-T wave abnormalities are noted in leads V5-V6 (↑); these are often associated with LVH. Although commonly called “strain,” they are actually repolarization abnormalities that result from chronic subendocardial ischemia. These abnormalities are often seen with hypertrophy as the blood flow to the subendocardium (the last part of the myocardium to receive blood and oxygen supply) is reduced as a result of the hypertrophied myocardium as well as an increased left ventricular end-diastolic pressure that often occurs with hypertrophy. The elevated left ventricular end-diastolic pressure causes a reduction in the pressure

gradient between the epicardium and endocardium, and hence a reduction in blood flow to the endocardium.

The usual ventricular rate for new-onset, untreated atrial fibrillation is between 140 and 180 bpm, which is the maximum rate at which the normal AV node can conduct impulses in the absence of autonomic nervous system inputs and without AV nodal blocking agents. A ventricular rate faster than 200 bpm (in the absence of preexcitation) suggests that there is a catecholamine effect that is enhancing AV nodal conduction (and may be the reason for the atrial fibrillation), such as hyperthyroidism, heart failure, or pulmonary embolism. In contrast, a ventricular rate of less than 100 bpm suggests intrinsic AV nodal disease, the effect of high vagal tone, or the use of an AV nodal blocking agent.

The ST-T wave changes noted are likely due to the increase of endocardial myocardial oxygen demand that is not met by the available oxygen supply. In the case presented, both rapid heart rates and LVH increase the demand for oxygen. When considering both of these factors, the patient can manifest signs and symptoms of cardiac ischemia even in the absence of underlying coronary artery disease and significant stenoses. Although the patient does not have any symptoms suggestive of coronary artery disease associated with ischemia, underlying coronary disease cannot be excluded by the information presented. Even though the ST-segment changes may be the result of demand ischemia, there should be an assessment for the presence of coronary artery disease after the patient is stabilized. This is especially the case if serial

continues

measurements of cardiac biomarkers (*ie*, troponin) are positive; along with the ST-segment changes, this would confirm the occurrence of demand ischemia and the strong possibility of coronary artery disease.

Immediate therapy should be aimed at reducing the ventricular response rate.

Rapid rate control can be achieved with AV nodal blocking agents such as β -blockers and calcium-channel blockers. Digoxin also blocks the AV node by enhancing vagal tone. However, its onset of action is delayed and it does not produce immediate slowing of the ventricular rate. As the patient does not have known reactive airway disease and is manifesting ECG changes that are concerning for myocardial ischemia, a β -blocker such as intravenous metoprolol followed by an oral dose for maintenance of rate control is a reasonable strategy. β -blockers tend to have less efficacy in patients with atrial fibrillation and a rapid ventricular rate in whom increased sympathetic activity may be present. This may be especially the case when the atrial fibrillation is of very recent onset, as there are acute hemodynamic changes that result in sympathetic activation. In patients who do not have other indications for β -blockers, non-dihydropyridine calcium-channel blockers (diltiazem and verapamil) are reasonable. Verapamil, which works primarily in the heart, tends to have more potent direct AV nodal blocking effects than diltiazem. However, it may cause more depression of cardiac inotropy. Diltiazem has more of a balanced effect on the heart (AV node) and the arterial circulation, causing vasodilatory effects that can offset the direct depressant effects on the AV node. It may also be

more likely to cause hypotension as a result of its peripheral arterial vasodilating effect.

If an initial trial of these agents causes hypotension but inadequate rate control, both digoxin and amiodarone are reasonable alternatives. Intravenous digoxin loading may have a measurable effect on the heart rate within 30 minutes, although peak effect may not be noted until 5 hours after the drug is administered. Oral loading is possible as well, although the effect will be even more gradual and delayed. The loading dose (generally 1.5 mg) used is independent of renal function, but maintenance doses are based on the estimated creatinine clearance (the maintenance dose is lowered in the presence of renal insufficiency). Serum levels can assist in guiding maintenance doses but have no role in loading.

Amiodarone is not used often for rate control in atrial fibrillation as its major benefit is maintenance of sinus rhythm. Therapy with amiodarone requires the administration of a loading dose, which takes weeks to months to achieve. A loading dose that can be initiated intravenously and continued orally is necessary as the drug is highly lipophilic, and lipid-enriched regions of the body must be saturated before serum levels rise. As a result there is a very large volume of distribution, and hence a prolonged time is required to achieve adequate serum levels. However, an initial bolus may have a measurable effect on heart rate, likely the result of β -blocking activity.

The risk for embolic stroke as a result of cardioversion in a patient with atrial fibrillation of unknown duration or for longer than 48 hours

who is not on an anticoagulant is 1% to 2%. In the patient who has been in atrial fibrillation for less than 48 hours, has been adequately anticoagulated for at least 4 weeks, or in whom transesophageal echocardiography (TEE) documents the absence of left atrial appendage thrombus prior to cardioversion, the risk for embolism is less than 1%. For the truly unstable patient, electrical cardioversion is an option, but again this should only be contemplated when heart rate slowing cannot be achieved and the patient is hemodynamically unstable. In this situation the risk for withholding therapy is greater than the potential thromboembolic risk.

Once the rate has been controlled, cardioversion may be considered. Numerous studies, of which AFFIRM (Atrial Fibrillation Follow-up Investigation of Rhythm Management) is the largest, have shown no difference in mortality, stroke, major bleeding, or heart failure between rate control with anticoagulation and restoration of sinus rhythm (rhythm control). AFFIRM suggested, however, that the subgroup of patients without heart failure who are older than 65 years of age may have had a mortality benefit with rate control rather than rhythm control. Nonetheless, many cardiologists and electrophysiologists make one attempt at rhythm control with direct current cardioversion. This may be at the time of atrial fibrillation diagnosis without preceding anticoagulation (TEE guided) or after 1 month of anticoagulation. In either case, a minimum of 1 month of anticoagulation after cardioversion to sinus rhythm is required because the risk for thromboembolism remains high. The observation has been that a combination of stunned and

inflamed atrial tissue, although contracting in sinus rhythm, confers an elevated thromboembolic risk for a period after electrical cardioversion.

The method of anticoagulation can vary from full-dose aspirin to warfarin (or a newer anticoagulant such as a direct thrombin inhibitor or a factor Xa inhibitor). The therapy chosen depends on the risk profile of the individual patient. The CHADS2 score is widely used to predict the risk for thromboembolic events in patients with atrial fibrillation. The score provides a point value to the features that confer elevated risk: congestive heart failure, hypertension, age 75 or older, diabetes, and secondary prevention (*ie*, history of ischemic cerebrovascular accident, transient ischemic attack, or other arterial embolism). Each feature is given a point score of 1, except secondary prevention, which is given a score of 2 (hence the “2” in “CHADS2”). The risk for stroke increases with increasing CHADS2 score, from 0.5% per 100 patient-years for a score of 1 to almost 7% per 100 patient-years for a score of 5 to 6. In general, patients with a CHADS2 score of 2 or higher should be on long-term warfarin therapy, maintaining an international normalized ratio (INR) of 2 to 3. For those who refuse to take warfarin or in whom there is a contraindication to this drug, one of the newer oral anticoagulants can be used, such as dabigatran (an oral direct thrombin inhibitor) or rivaroxaban, apixaban, or edoxaban (oral factor Xa inhibitors). Therapy with aspirin and clopidogrel is an alternative. Therapy with full-dose aspirin (325 mg/day) rather than warfarin is an alternative for patients with a CHADS2 score of less than 2.

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Atrial fibrillation can be characterized by the duration of the arrhythmia. Intermittent atrial fibrillation (previously termed paroxysmal atrial fibrillation) is defined as self-limiting episodes. Persistent atrial fibrillation is atrial fibrillation that continues until chemical or electrical cardioversion is performed. Atrial fibrillation is considered permanent (previously termed chronic) when it is either resistant to cardioversion or a decision is made to allow the patient to remain in atrial fibrillation.

In this case, immediate rate control with β -blockade is reasonable. Given the patient's young age, it would be reasonable to attempt cardioversion to restore sinus rhythm. This is best performed after 4 weeks of adequate anticoagulation (INR 2–3). If the patient's preference is more

immediate cardioversion or if immediate cardioversion is necessary for hemodynamic control, TEE would be required to exclude a left atrial appendage thrombus prior to reversion. Once reverted (either immediately or after 4 weeks of anticoagulation), 4 weeks of anticoagulation would be required.

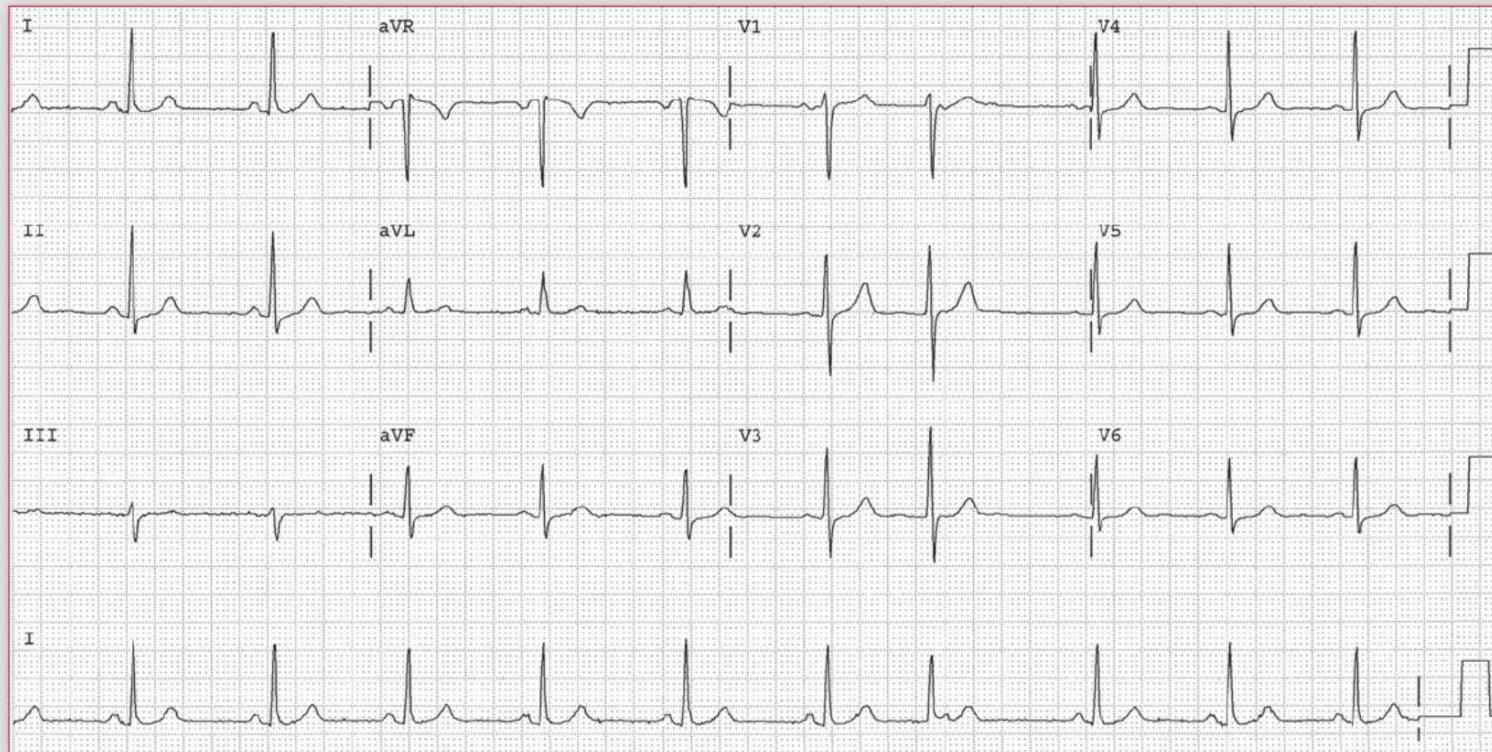
It is possible that in this patient, obesity and sleep apnea are the likely triggers of atrial fibrillation. However, a search for other modifiable causes (eg, hyperthyroidism, structural or valvular heart disease, signs of elevated right heart pressures) should be sought. If the patient prefers to remain in atrial fibrillation with rate control, he would require some form of anticoagulation. Since his CHADS2 score is low (*ie*, 1 as the only risk factor is hypertension), aspirin therapy alone would be acceptable. ■

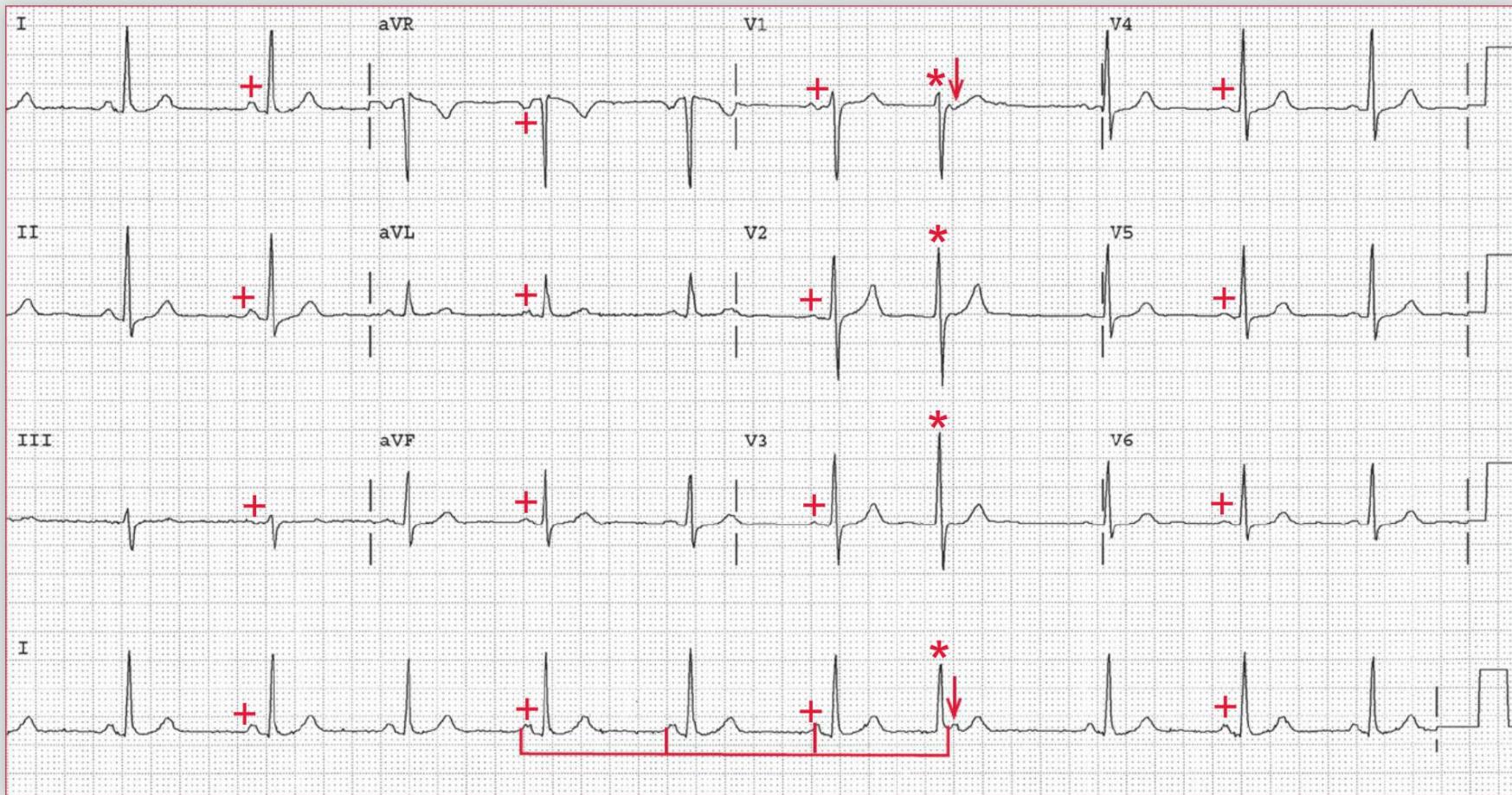
A 27-year-old man presents to his primary care physician with complaints of nocturnal palpitations that began with an indolent onset several weeks ago when he noted the sensation of irregular heartbeats when lying down to sleep at night. He denies associated symptoms of any kind and generally does not experience symptoms during the day. The sensation is so unnerving that he has had trouble sleeping. He does not wake with these symptoms, however.

The patient is otherwise active and without physical limitations. He denies changes in his diet, specifically regarding caffeine and chocolate, and denies new exposure to over-the-counter medications. He has not had recent exposure to tick-endemic regions. His medical and surgical history are unremarkable. He has no family history of cardiac disease. His physical exam is normal, but some pressured speech is noted. An ECG is obtained.

Based on the ECG, what is the cause of his symptoms?

What further testing, if any, is needed?





ECG 30 Analysis: Normal sinus rhythm, premature junctional complex

The rhythm is regular at a rate of 62 bpm. The QRS complex has a normal duration (0.08 sec), morphology, and axis (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/390 msec). There is a P wave (+) before each QRS complex with a normal P-wave morphology (positive in leads I, II, aVF, and V5-V6) and a stable PR interval (0.16 sec). Therefore, this is a normal sinus rhythm. There is a single premature complex (*) that has the same QRS complex morphology as the sinus complexes, but there is no P wave preceding this QRS complex. This is a premature junctional complex (PJC). There is, however, a P wave (↓) following the premature QRS complex; this P wave is the on-time sinus P wave as it has the same PP interval as all the other PP intervals (◻).

A PJC is identified by a premature QRS complex that has a morphology similar to that of the sinus complex but without a preceding P wave. There may or may not be a P wave following the premature QRS complex. If there is a P wave after the PJC that is inverted, often with a shorter PP interval compared with the sinus PP interval, this is referred to as a retrograde P wave and is due to ventriculoatrial conduction.

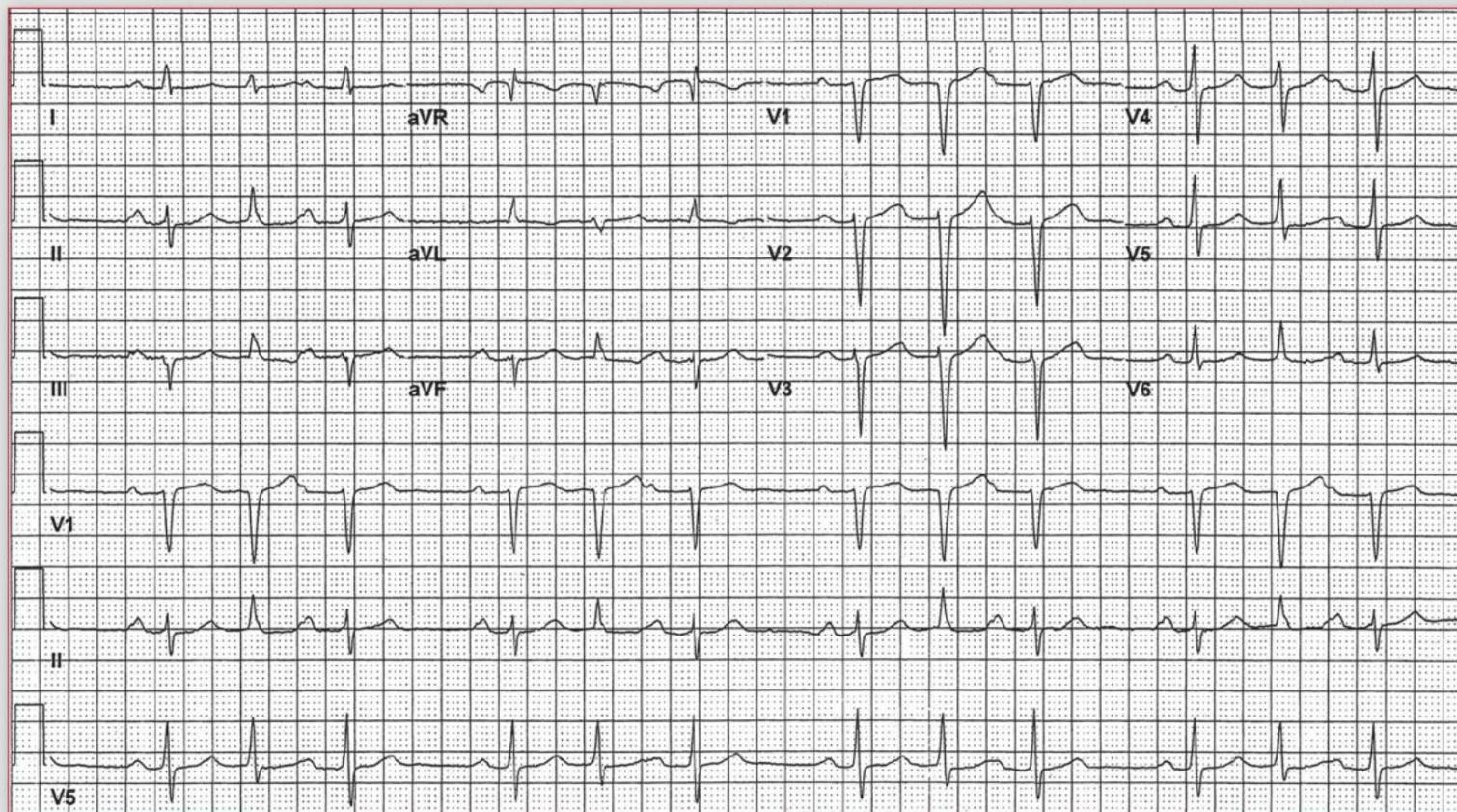
If there are several PJCs, the RP interval will be stable. Alternatively, the P wave following the PJC may be an on-time sinus P wave. In this situation, the P wave will have the same PP interval and morphology as the P waves that precede the other QRS complexes (*ie*, it is upright in leads I, II, aVF, and V4-V6). In this case the P wave following the PJC is indeed the on-time sinus P wave.

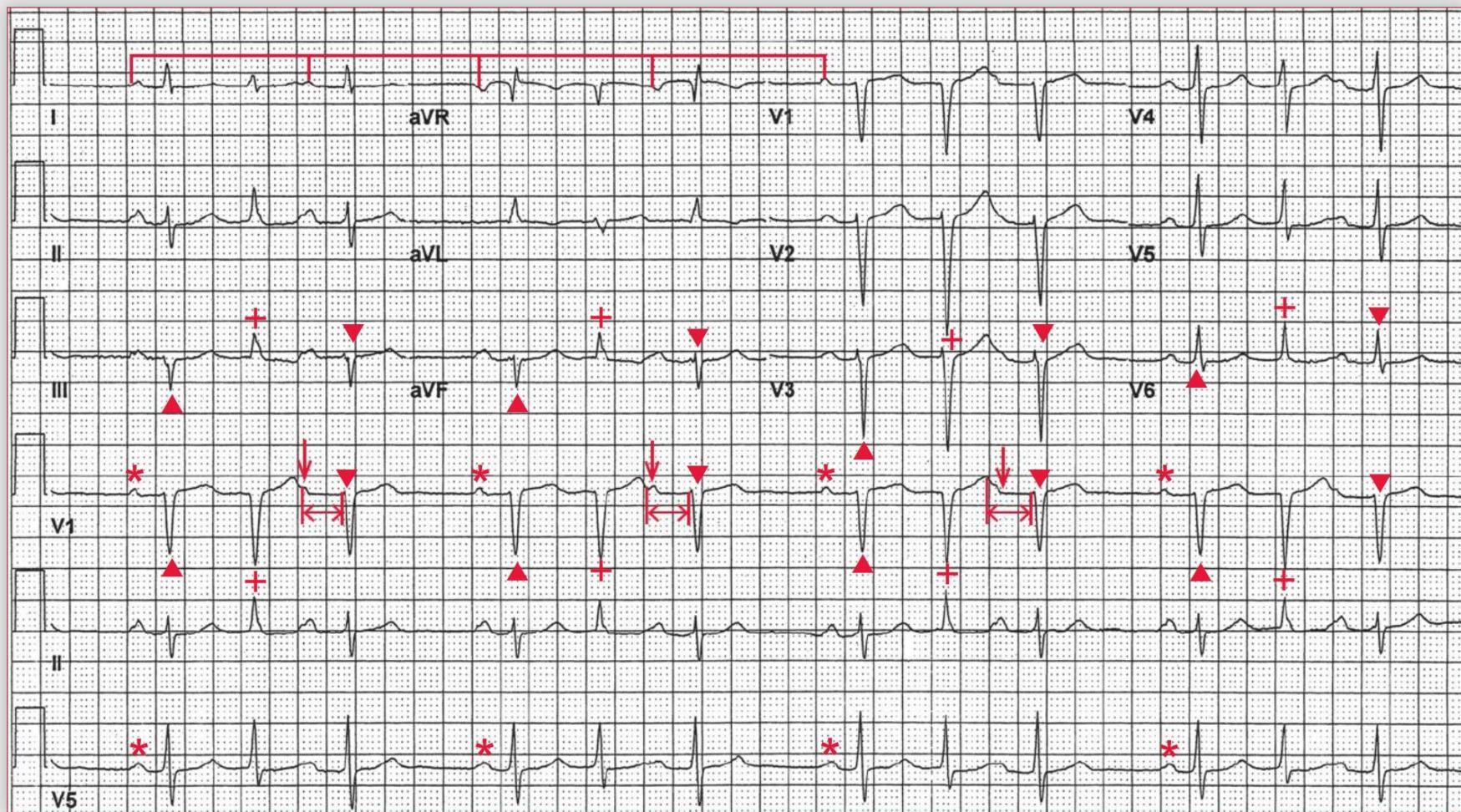
Palpitations are a nonspecific complaint that may have a myriad of cardiac (often conduction abnormalities or arrhythmia) and noncardiac causes. Palpitations due to a cardiac cause are often benign and may result from environmental or dietary exposures such as caffeine or stimulants, drug toxicities (*eg*, digitalis), or underlying thyroid disease, or they may have a more serious pathology such as potentially serious arrhythmia, incipient cardiac conduction system disease, or cardiomyopathy (ischemic or non-ischemic). In this case, given the patient's age and negative review of systems, the likely cause for the palpitations is benign PJCs. However, thyroid function should be assessed as insomnia, pressured speech, and supraventricular arrhythmias may be the result of hyperthyroidism. ■

Notes

A 72-year-old woman presents to her cardiologist with the sensation of her heart “skipping beats.” She states that the symptoms started about 2 weeks prior to being seen. Upon further review, she recalls that she started a new anti-hypertensive medication at the request of her primary care physician a few days before symptom onset. She does not recall the name of the medication. Review of symptoms is also notable for increased nocturia over the past several weeks. Her physical exam is unremarkable except for a regularly irregular radial pulse. An ECG is obtained.

What is the cause of the patient’s symptoms?
What is the likely underlying etiology?
Is any therapy necessary?





ECG 31 Analysis: Sinus bradycardia, left axis, first-degree AV block, interpolated premature junctional complexes in a trigeminal pattern

There is a regularly irregular rhythm with group beating; there are groups of three QRS complexes that have a repeating pattern. The first complex of each group (▲) has a preceding P wave (*) with a stable PR interval (0.26 sec) that is prolonged. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus complex with a first-degree AV block or prolonged AV conduction. The QRS complex has a normal duration (0.10 sec) and a left axis of about -30° (positive QRS complex in lead I, negative QRS complex in lead aVF, and isoelectric QRS complex in lead II). The second QRS complex (+) of the three has the same duration (0.10 sec) and the same morphology; however, it has a different axis, that is, the axis is normal, between 0° and $+90^\circ$ (positive QRS complex in leads I, II, and aVF). In addition, this QRS complex has a different amplitude compared with the first and third QRS complexes, as seen in leads V1-V5. There is no P wave preceding this complex. Hence these are premature junctional complexes (PJC). The third QRS complex (▼) that follows the PJC has the same morphology as the first. There is also an on-time P wave (↓) before these complexes; the PP interval is constant throughout (⊤). The P wave has the same morphology and axis as the sinus P wave that precedes the first QRS complex. The third of the three QRS complexes is also an on-time sinus complex. Hence there is a sinus rate of 50 bpm. However, the PR interval (\leftrightarrow) of this second sinus complex is longer (0.30 sec) than the PR interval of the first sinus complex. This is the result of retrograde concealed conduction, which occurs when the PJC results in partial retrograde penetration and depolarization of

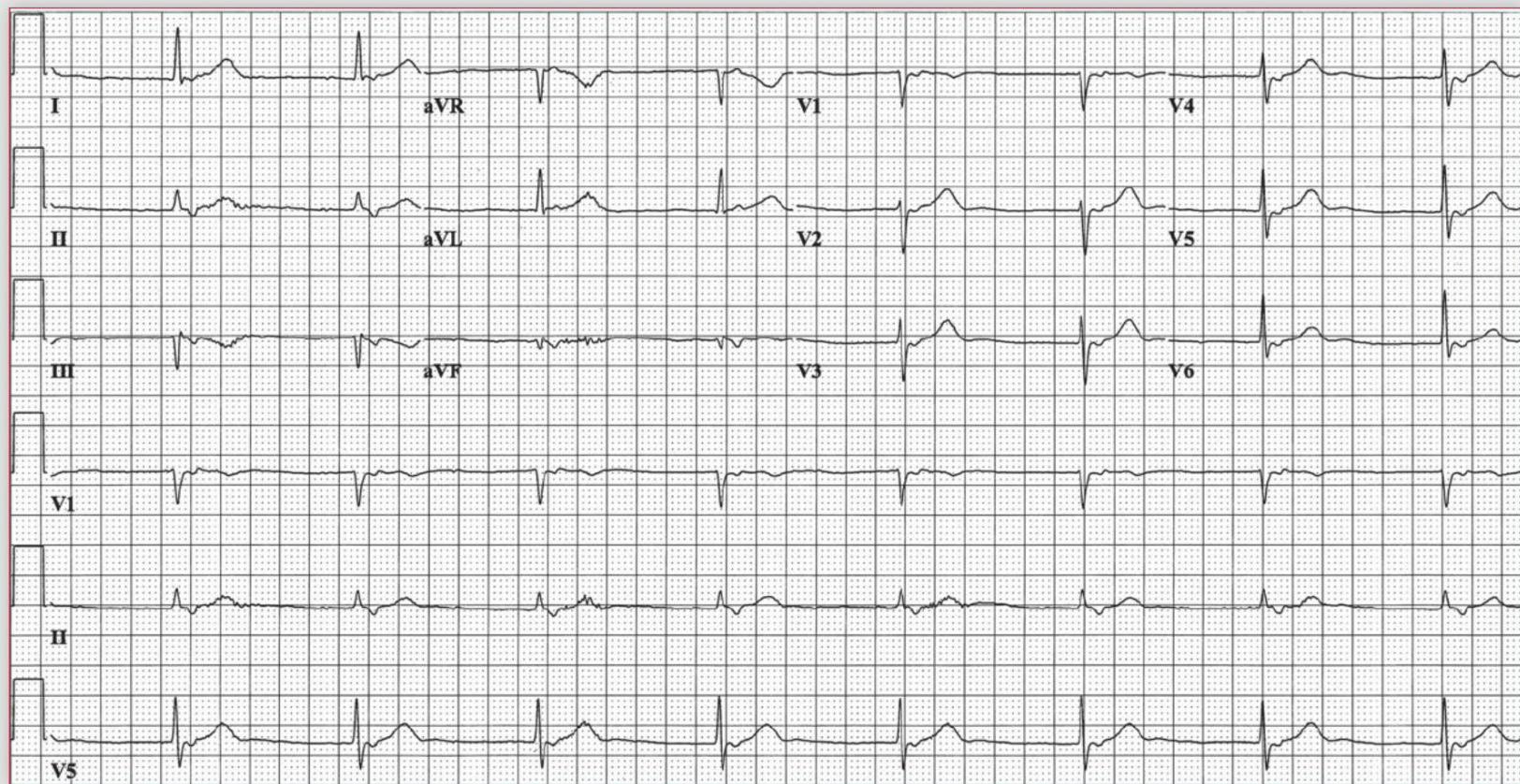
the AV node and causes a partial prolongation of AV nodal refractoriness. Therefore, the subsequent on-time sinus impulse can conduct through the AV node, but the rate of conduction is slower as a result of the partial depolarization by the PJC. Since every third complex is a PJC, this is junctional trigeminy. In addition, the PJC does not result in any pause or alteration of the PP interval; hence these are termed interpolated PJC. Noted is that the PJC have an axis and amplitude that are different than those of the sinus QRS complexes. This is commonly seen with junctional complexes and is due to the fact that the junctional complex results from an ectopic focus in the AV junction that generates an impulse that enters the bundle of His at a different location compared with an impulse that is generated in the atrium and is conducted through the AV node. The conduction through the His-Purkinje system (which is a series of tracts) may, therefore, be different, accounting for the differences in QRS axis and/or amplitude. The QT/QTc intervals are normal (440/400 msec).

PJC may be caused by a wide range of underlying conditions. Similar to premature atrial complexes, they are generally benign and idiopathic, although they may be related to electrolyte abnormalities, digitalis toxicity, incipient cardiomyopathy (ischemic or otherwise), or thyroid dysfunction. In this patient, who has recently started an anti-hypertensive medication, the possibility of hypokalemia due to diuretic therapy should be evaluated. ■

Core Case 32

A 52-year-old man is seen in clinic by his cardiologist for a routine assessment. His medical history includes a prior myocardial infarction and paroxysmal atrial fibrillation. He has been well without new complaints. On exam, the cardiologist is puzzled by the

ECG 32A



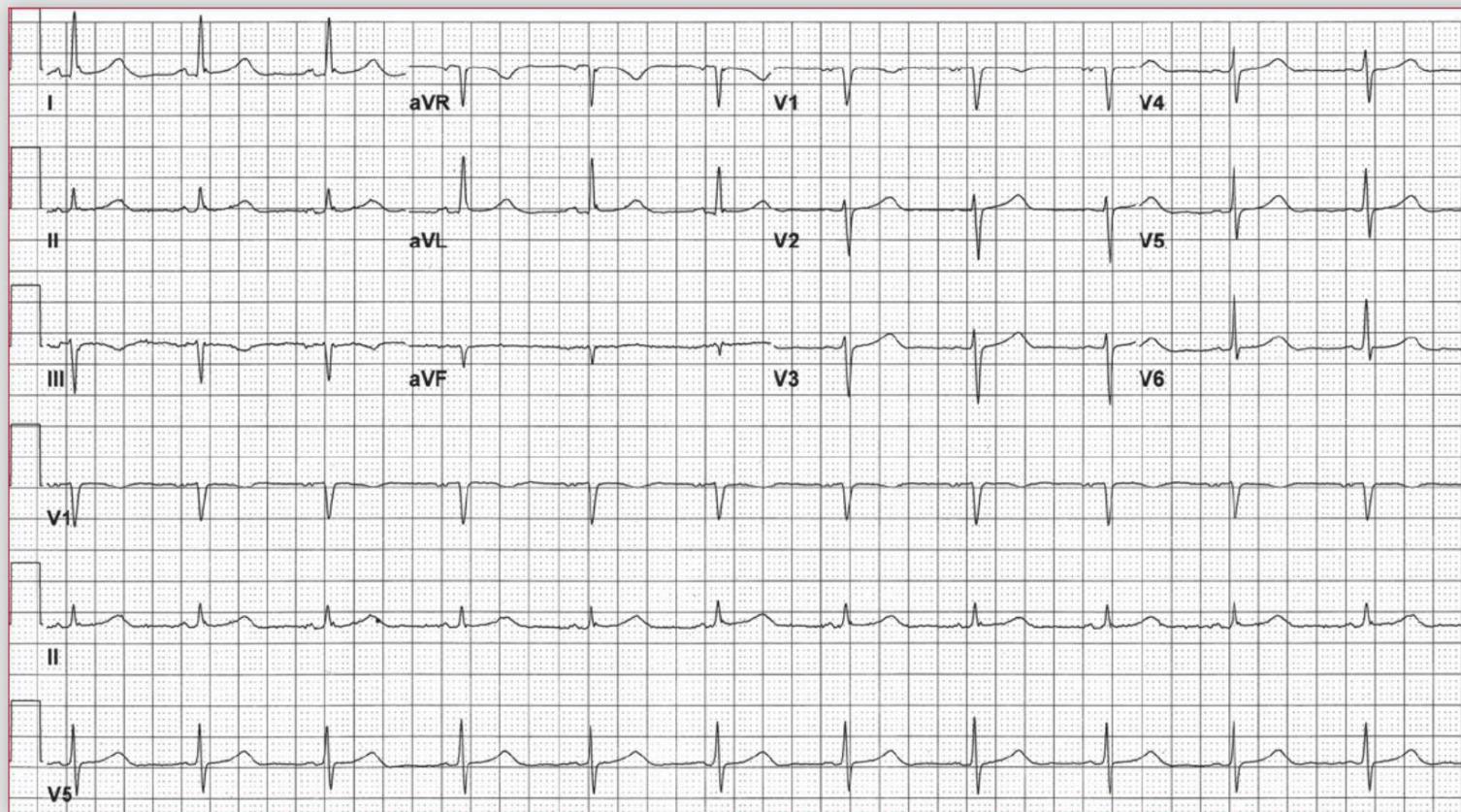
patient's cardiac auscultatory exam. Initially, the heart sounds were irregular, but while listening the heart sounds have become regular. The cardiologist obtains an ECG (32A). Several minutes later a second ECG is obtained (ECG 32B).

What abnormalities are apparent in ECG 32A?

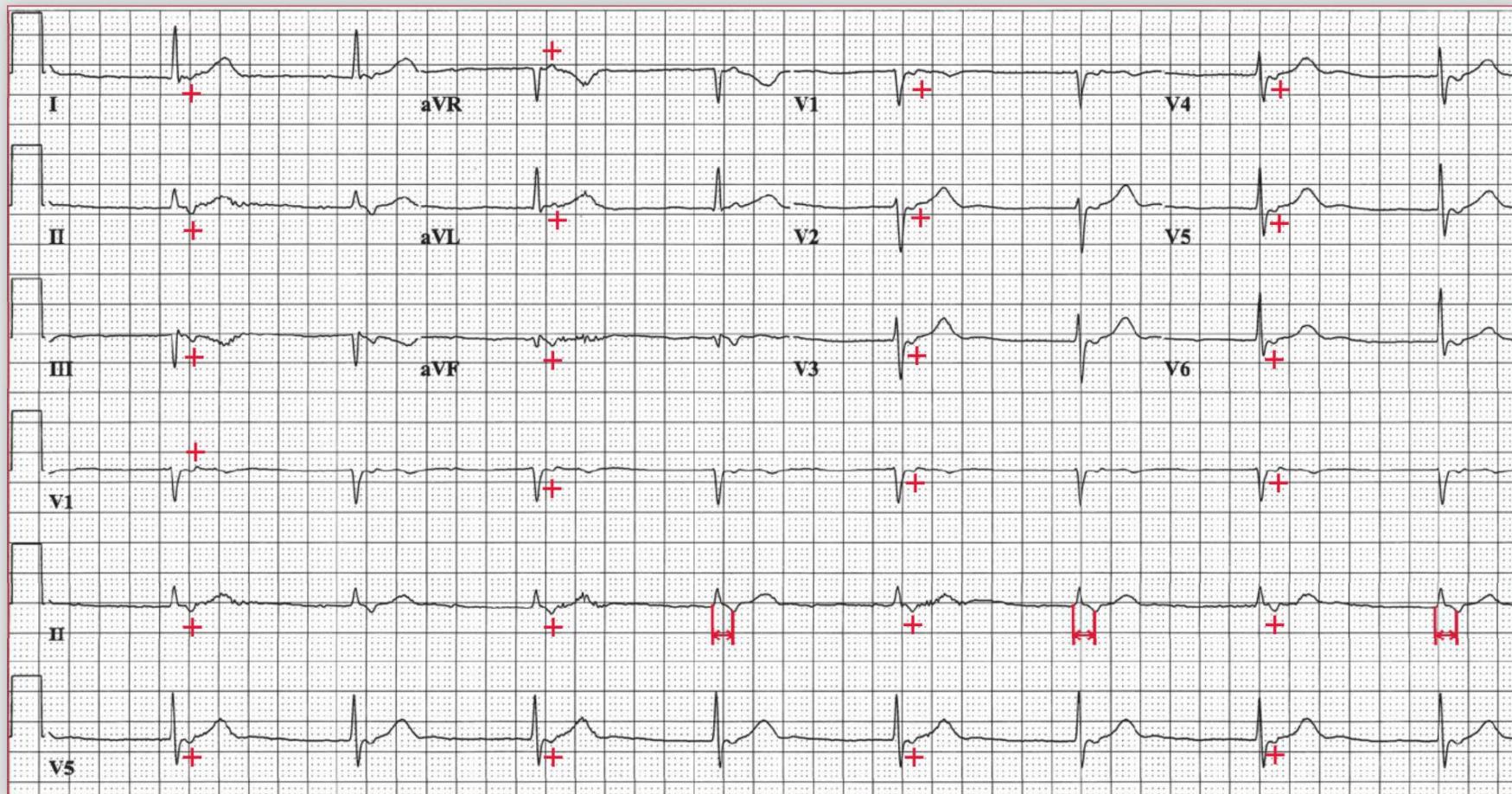
What is the rhythm in ECG 32B?

After reviewing both ECGs, can you discern what the cardiologist noted on exam that prompted the ECG?

ECG 32B



Podrid's Real-World ECGs



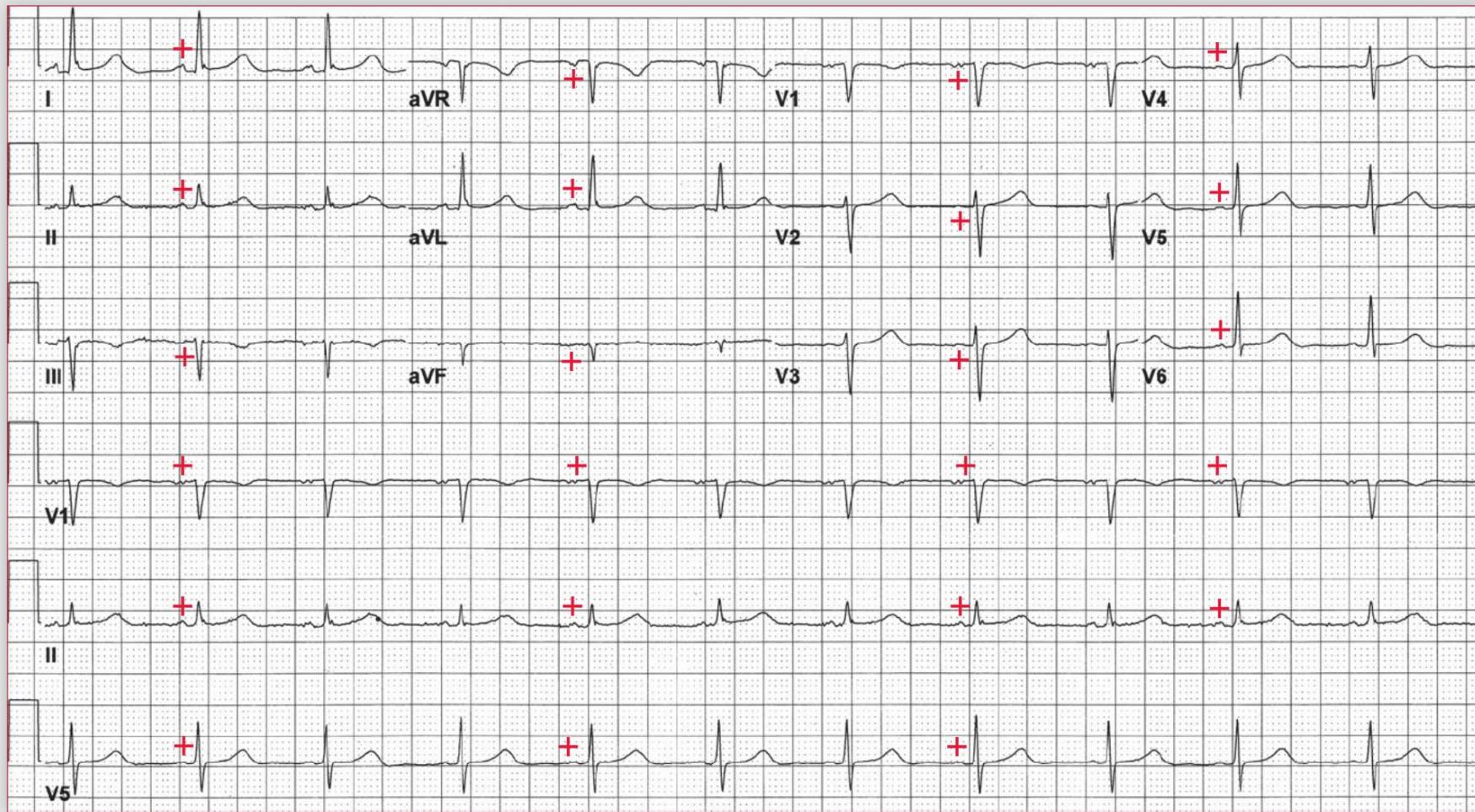
ECG 32A Analysis: Junctional rhythm, retrograde (ventriculoatrial) conduction, retrograde P wave, left axis

ECG 32A shows a regular rhythm at a rate of 50 bpm. The QRS complex is of normal duration (0.08 sec) and the axis is leftward (physiologic), between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (470/420 msec). There are no P waves before any QRS complex; hence this is a junctional rhythm. In most of the leads negative deflections are noted within the ST segment (+) before the T waves. These waveforms are narrow and have the peaked appearance of a P wave. They are in fact negative or retrograde P waves with a fixed RP interval (0.12 sec) (↔), and hence there is retrograde atrial activity due to ventriculoatrial conduction from the junctional beats.

Based on the auscultatory findings, it is possible that the patient was initially in atrial fibrillation that abruptly terminated into a regular rhythm. In this case, the appreciation on exam of a rhythm that was initially irregular and then regular prompted the cardiologist to obtain an ECG. In a patient with paroxysmal atrial fibrillation, termination of

the arrhythmia should lead to resumption of sinus rhythm. However, in this patient the absence of fibrillatory waves with a regular rhythm indicates that atrial fibrillation is no longer present. Additionally, the absence of sinus P waves suggests that the fibrillation has self-terminated but sinus node activity has failed to be restored. Hence there is probably sinus arrest with a junctional escape. This is the manifestation of sinus node dysfunction, or sick sinus syndrome (*ie*, a tachycardia-bradycardia syndrome). With atrial fibrillation there is depression of sinus node activity. However, if there is sinus node dysfunction, sinus node activity fails to be restored appropriately when the atrial fibrillation terminates abruptly. Sick sinus syndrome is often associated with an abnormality of the AV node (as the syndrome often affects both pacemaker structures). In this case, the patient has intact and normal AV nodal function. However, if there were AV nodal disease as well, it is possible that the offset pause would have been associated with a period of asystole.

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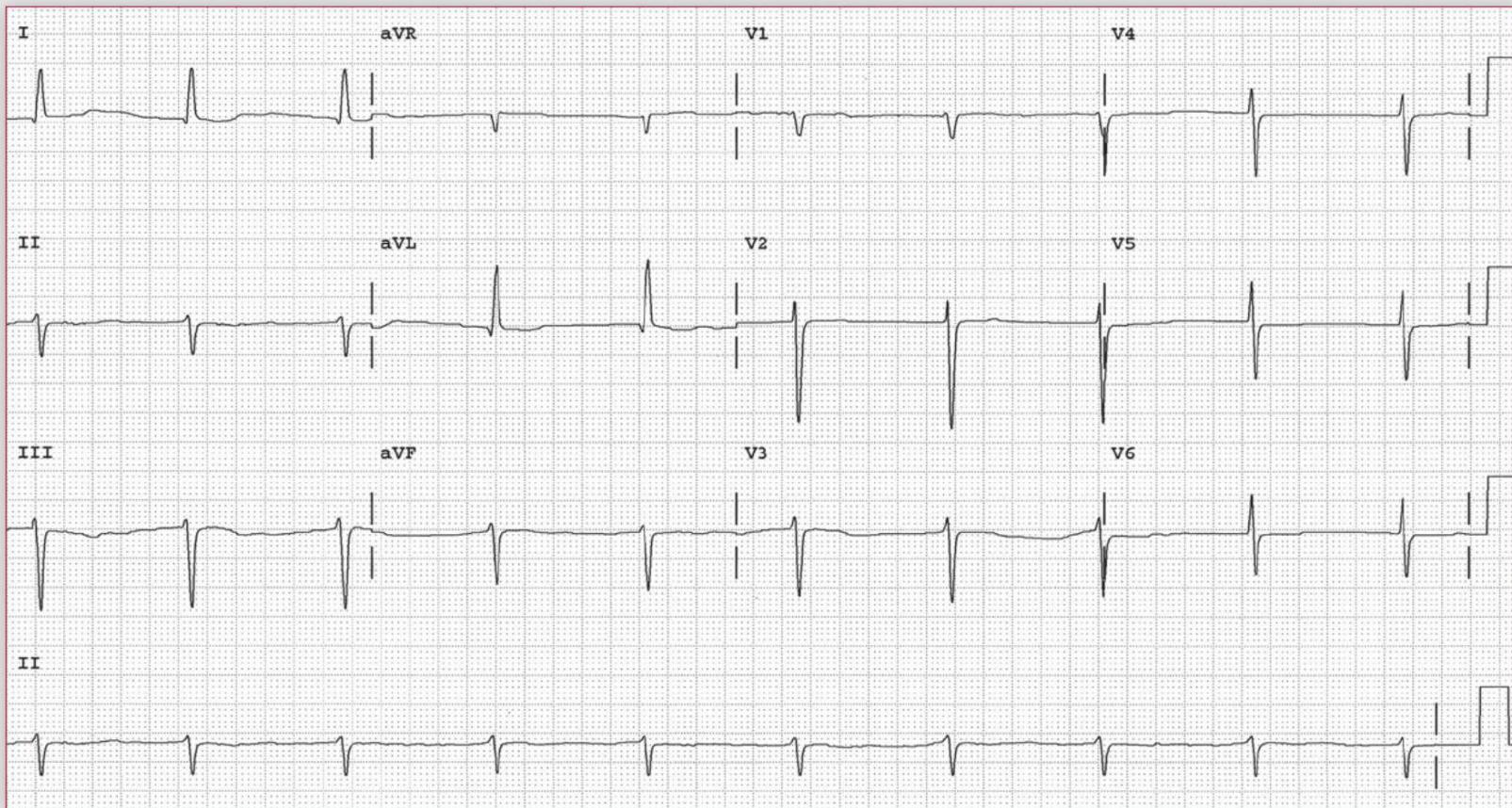
ECG 32B Analysis: Normal sinus rhythm, left axis

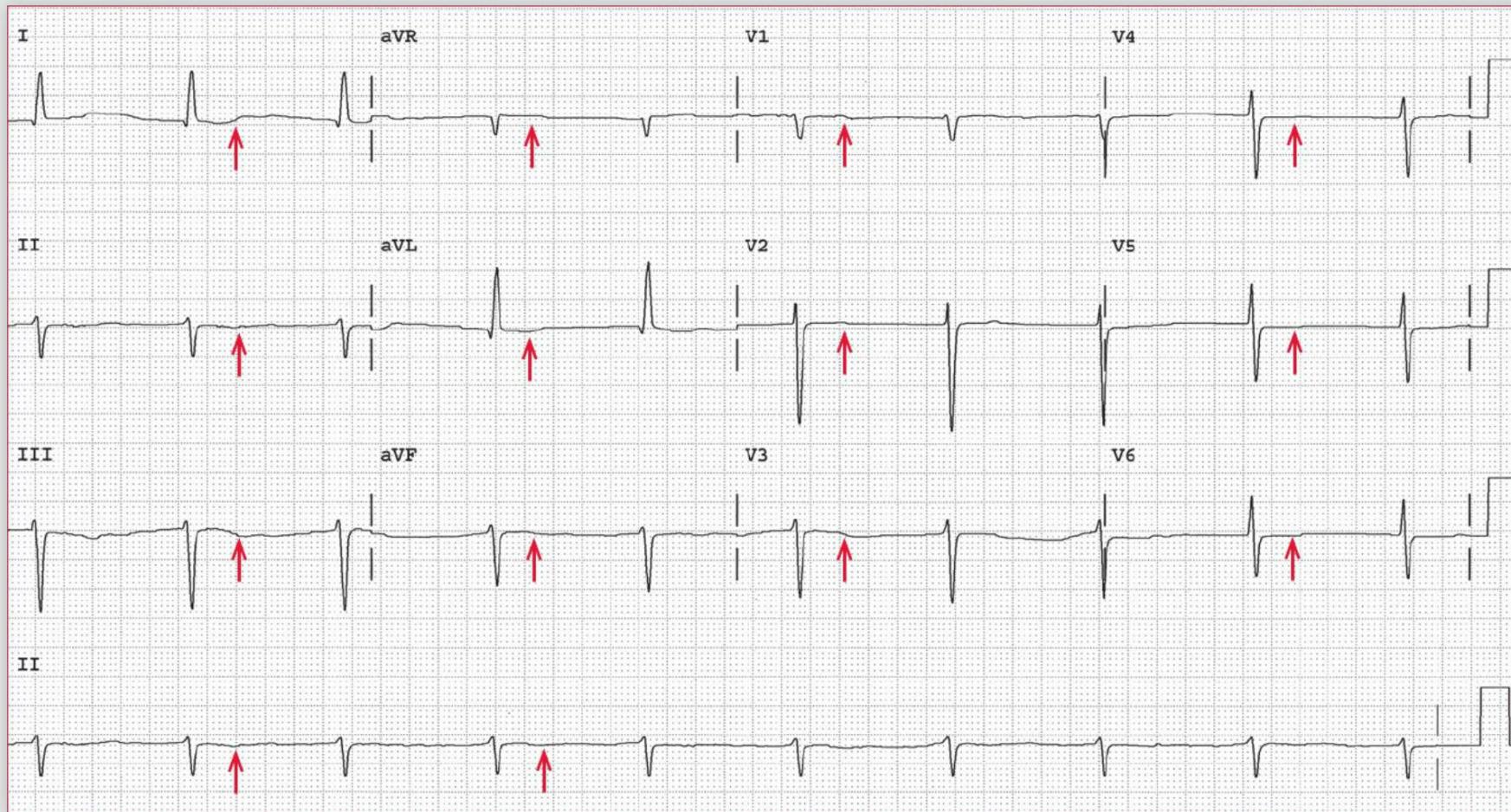
ECG 32B shows a regular rhythm at a rate of 70 bpm. There is a P wave before each QRS complex, and it is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration, morphology, and axis are identical to what is seen in ECG 32A. The QT/QTc intervals are normal. There are no retrograde P waves noted as this is now a normal sinus rhythm, reflecting restoration of sinus node activity. ■

Notes

A 57-year-old man with a pacemaker is playing recreational baseball for his local town team. A line drive hits him in the left shoulder at the site of his pacemaker generator. He loses consciousness for several seconds then spontaneously regains consciousness. He is taken to the local emergency department. On arrival, he is awake and alert, complaining only of pain at the left shoulder. He states that he has had the pacemaker for many years and that the rate is set at 60 bpm. A chest radiograph shows a pacemaker generator with two leads (right atrial and right ventricular). An ECG is obtained.

Based on the presentation, ECG, and chest radiograph, what is the likely cause of this patient's loss of consciousness?





ECG 33 Analysis: Junctional rhythm, left anterior fascicular block, clockwise rotation (poor R-wave progression and late transition), nonspecific ST-T wave abnormalities

There is a regular narrow QRS complex rhythm at a rate of 58 bpm. There are no P waves before or after any of the QRS complexes. Hence this is a junctional rhythm. The QRS complexes are of normal duration (0.08 sec) and morphology with poor R-wave progression in leads V1-V3 and late transition (*ie*, clockwise rotation of the heart in the horizontal plane). This is determined by imagining the heart as viewed from under the diaphragm, with the right ventricle in front and the left ventricle to the left side. With clockwise rotation, left ventricular forces appear late in the precordial leads as the left ventricle is electrically rotated to the back and right ventricular forces extend toward the left.

The axis in the frontal plane is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology), and hence this is a left anterior fascicular block. In addition, there are diffuse nonspecific T-wave changes (\uparrow) with T-wave flattening in all leads. The QT/QTC intervals are normal (460/450 msec).

This patient likely had a pacemaker implanted for sinus node arrest or sick sinus syndrome as his underlying rhythm is now junctional with the absence of any atrial activity. It can be assumed that he had significant junctional bradycardia that was symptomatic and warranted device implantation. Therefore, the possible abrupt loss of pacemaker

function after the device was struck by a baseball would unmask a significantly bradycardic underlying rhythm that resulted in syncope. It is probable that the rate of the junctional rhythm increased by the time the ECG was obtained.

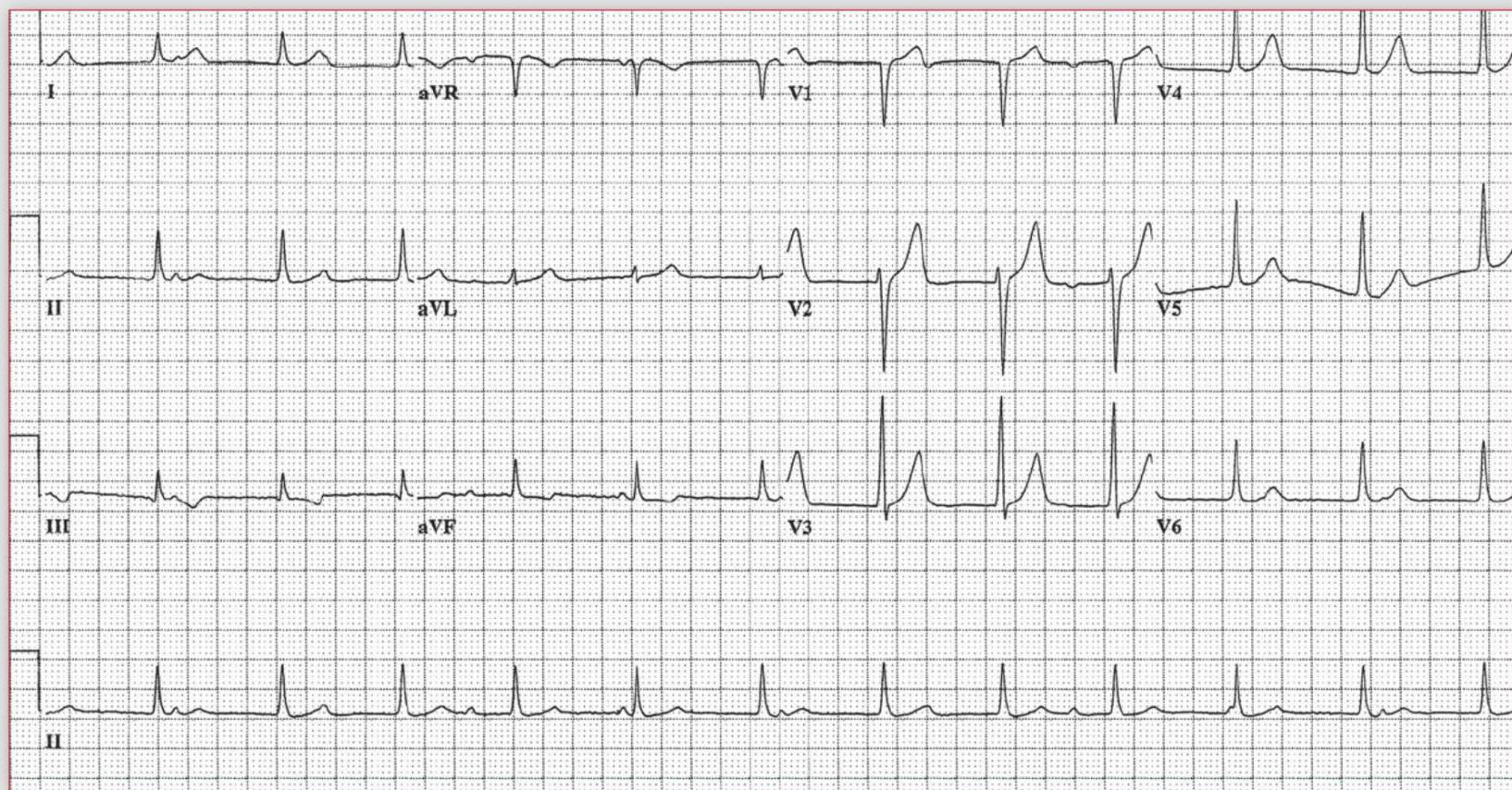
The chest radiograph demonstrated that the patient has a dual-chamber pacemaker, with one lead terminating in the right atrium and the other in the right ventricle. Given the absence of both pacemaker activity and P waves on the ECG, it can be assumed that the trauma to the device disrupted the function of both leads. If the atrial lead had fractured from the force of the blow, a bradycardic rhythm would result in the occurrence of a ventricular paced rhythm at the backup or lower rate limit of the pacemaker. This would have prevented the syncope. If the ventricular lead had been disrupted and there was an absence of native AV conduction, atrial paced P waves would be seen, but the patient would be asystolic until a spontaneous ventricular rhythm was restored. If AV conduction was intact, the patient would likely have been unaware of any problem until his next pacemaker interrogation. The fact that there is no evidence of atrial or ventricular pacemaker lead function suggests that either the generator has been damaged or that both leads have been dislodged from the generator at their point of origin. Pacemaker interrogation will allow the final diagnosis to be made. ■

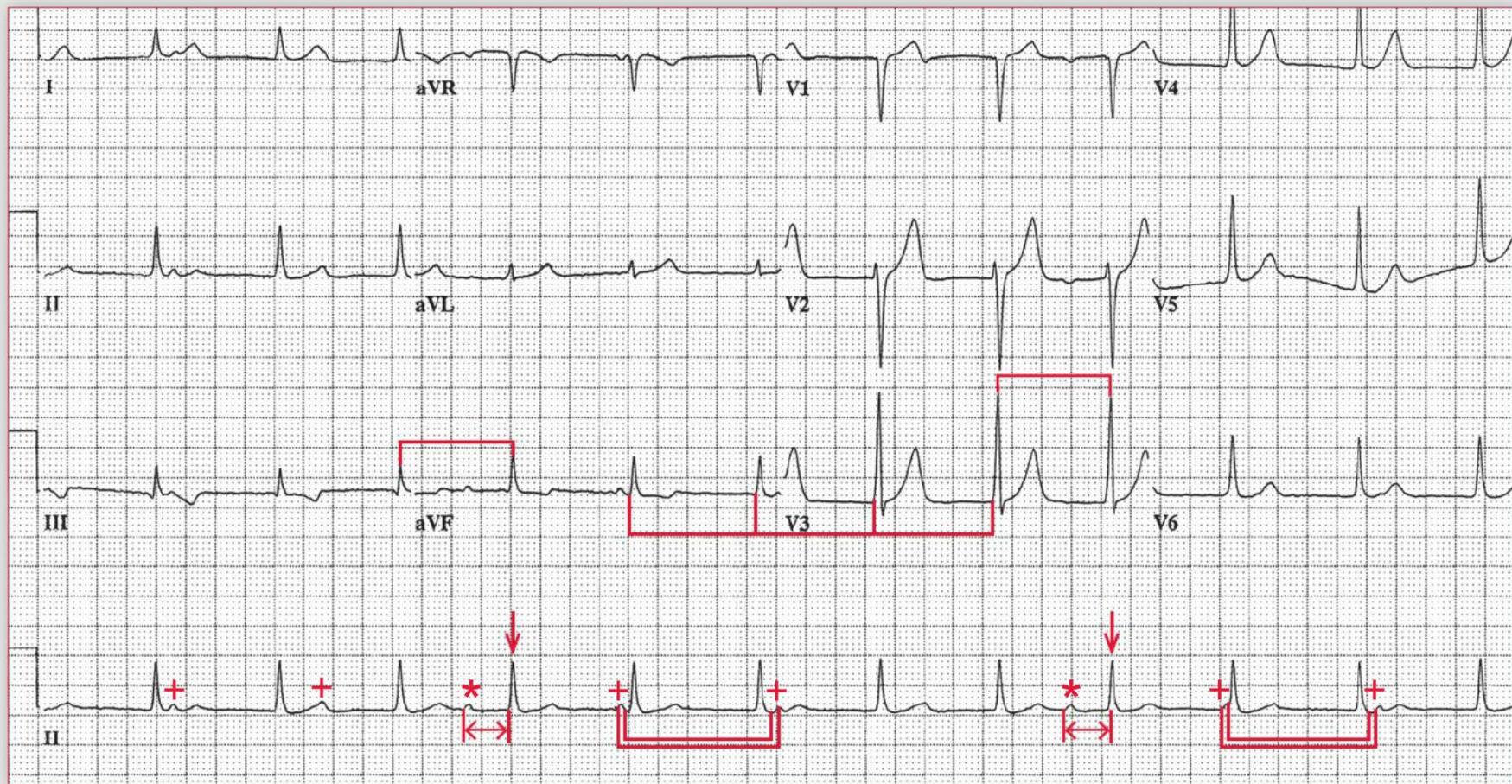
Notes

A 62-year-old woman is postoperative day 1 following coronary artery bypass grafting. Her history is notable for first-degree AV block in addition to coronary artery disease.

The consulting cardiologist states that the temporary pacemaker electrode can be removed based on the ECG obtained that morning. The cardiothoracic surgeon objects, stating that the patient has not documented intact AV conduction as of yet.

Based on the ECG, with whom do you agree?





ECG 34 Analysis: Sinus bradycardia with accelerated junctional rhythm, captured complex with first-degree AV block

The rhythm is regular (RR intervals = 0.84 sec) (◻) except for the fourth and ninth QRS complexes (↓), which are slightly premature (RR intervals = 0.72 sec) (◻). Both of these complexes are preceded by a P wave (*), and the PR interval (↔) is the same (0.32 sec). There is variability of the other PR intervals, without any association with the QRS complexes. Some of the QRS complexes do not have any associated P wave, while others have a P wave before or after them (+). There is, therefore, AV dissociation. The P waves are positive in leads I, II, aVF, and V4-V6; hence there is an underlying sinus rhythm. The sinus rate is stable at 58 bpm (◻), while the rate of the QRS complexes is 72 bpm. The QT/QTc intervals are normal (360/390 msec). As the atrial rate is slower than the ventricular rate, this is an accelerated lower pacemaker. Since the QRS complexes have a normal duration (0.08 sec), morphology, and axis, between 0° and +90° (positive QRS complex in leads I and aVF), the accelerated rhythm is junctional. Hence this is sinus bradycardia with AV dissociation and an accelerated junctional rhythm. The two early QRS complexes (↓), which have the same PR interval, are responding to the atrial activity before them, and hence these are two captured sinus beats. They are the result of P waves

that occur at a time when AV nodal conduction can occur and hence they capture the ventricle. However, they do not alter the underlying accelerated junctional rhythm.

There are two etiologies for AV dissociation. The first is complete, or third-degree, AV block. In this situation the atrial rate is faster than the rate of the QRS complexes as the QRS complexes are due to an escape rhythm. The escape rhythm may be either junctional or ventricular, and this is based on the QRS complex morphology and not the QRS complex rate. The presence of complete heart block that is permanent is generally an indication for a permanent pacemaker.

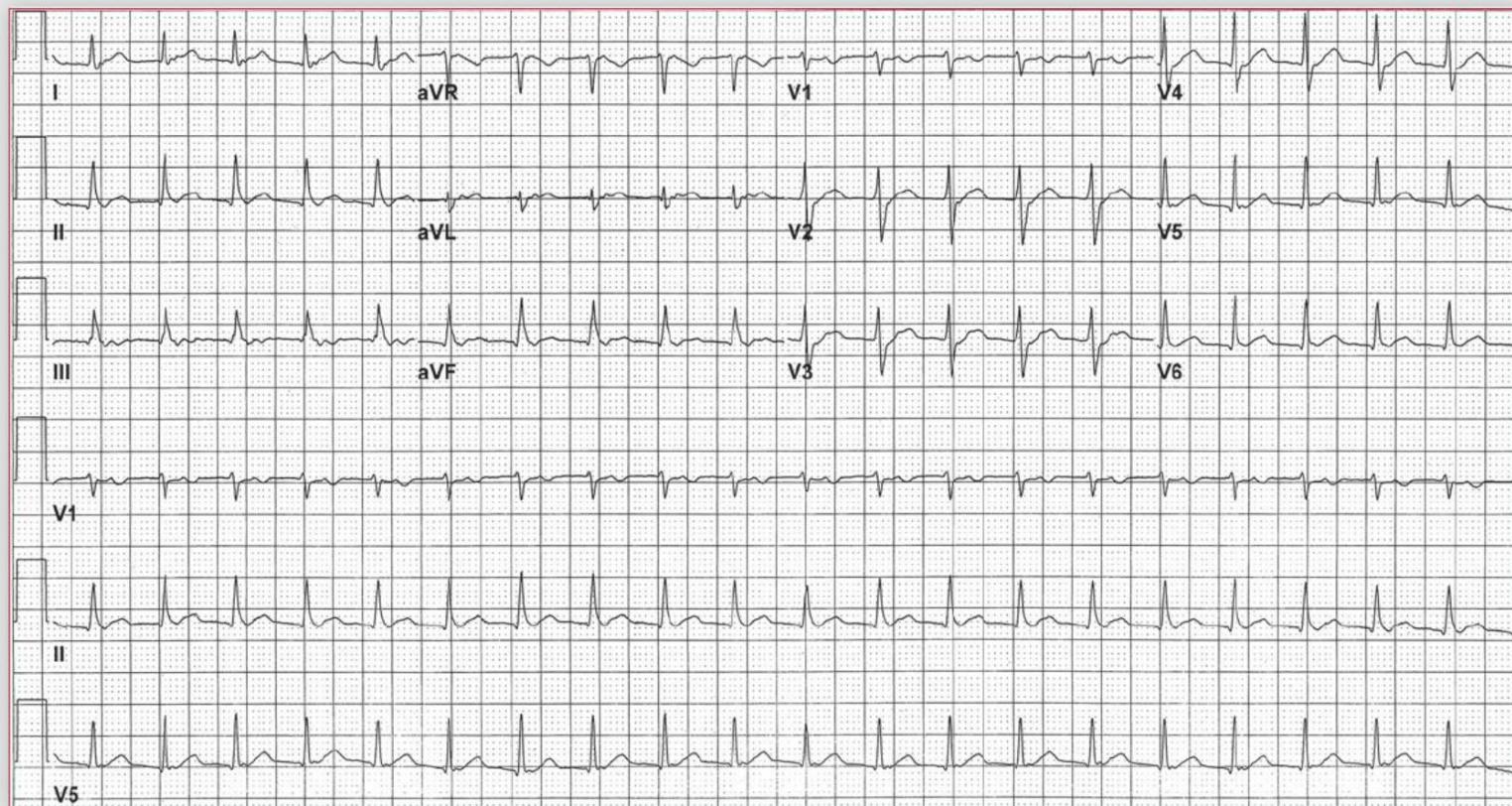
The second etiology is an accelerated lower pacemaker focus, either junctional or ventricular. In this situation, the atrial rate is slower than the rate of the QRS complexes. There is no problem with AV conduction, but rather a slower pacemaker has become accelerated to a rate faster than the sinus rate. Hence a pacemaker is not necessary as there is intact AV conduction, as indicated here by the presence of the two captured complexes. ■

Core Case 35

A 32-year-old woman presents to her primary doctor's urgent care clinic with complaints of profound anxiety. She states that she has had episodes of anxiety, diaphoresis, and "a racing heart" for the past week. She does not have a history of anxiety disorder. She has also had several bouts of diarrhea over the past 2 to 3 days.

Her physician places his hand on her radial pulse and promptly orders an ECG (35A). A physical exam, conducted

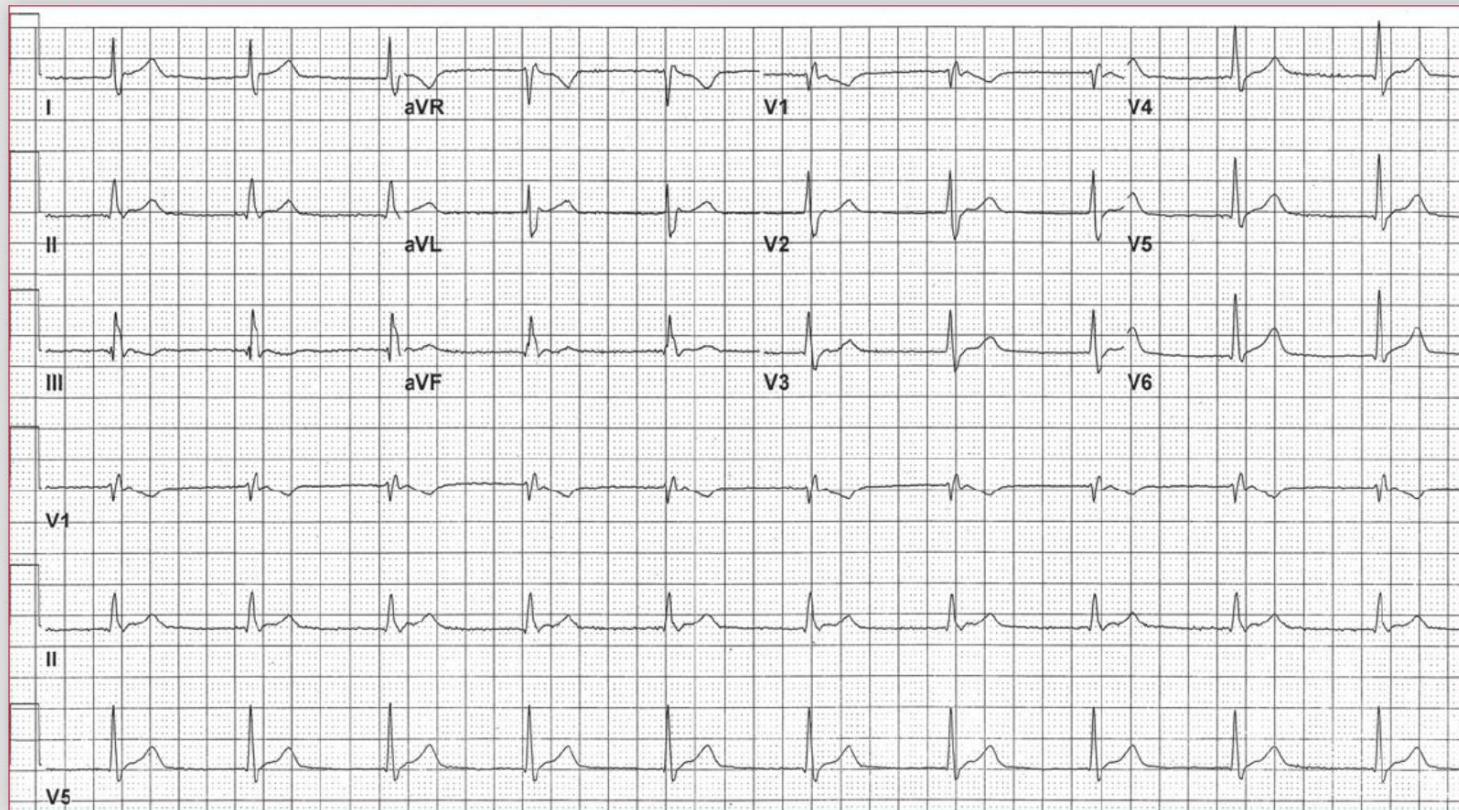
ECG 35A



while waiting for the ECG, reveals a temperature of 101.4°F, a heart rate of 130 bpm, pressured speech, and mild facial flushing. The patient is transferred to the local emergency department.

Upon arrival in the emergency department, intravenous β -blockers are administered, after which a second ECG is obtained (ECG 35B).

ECG 35B

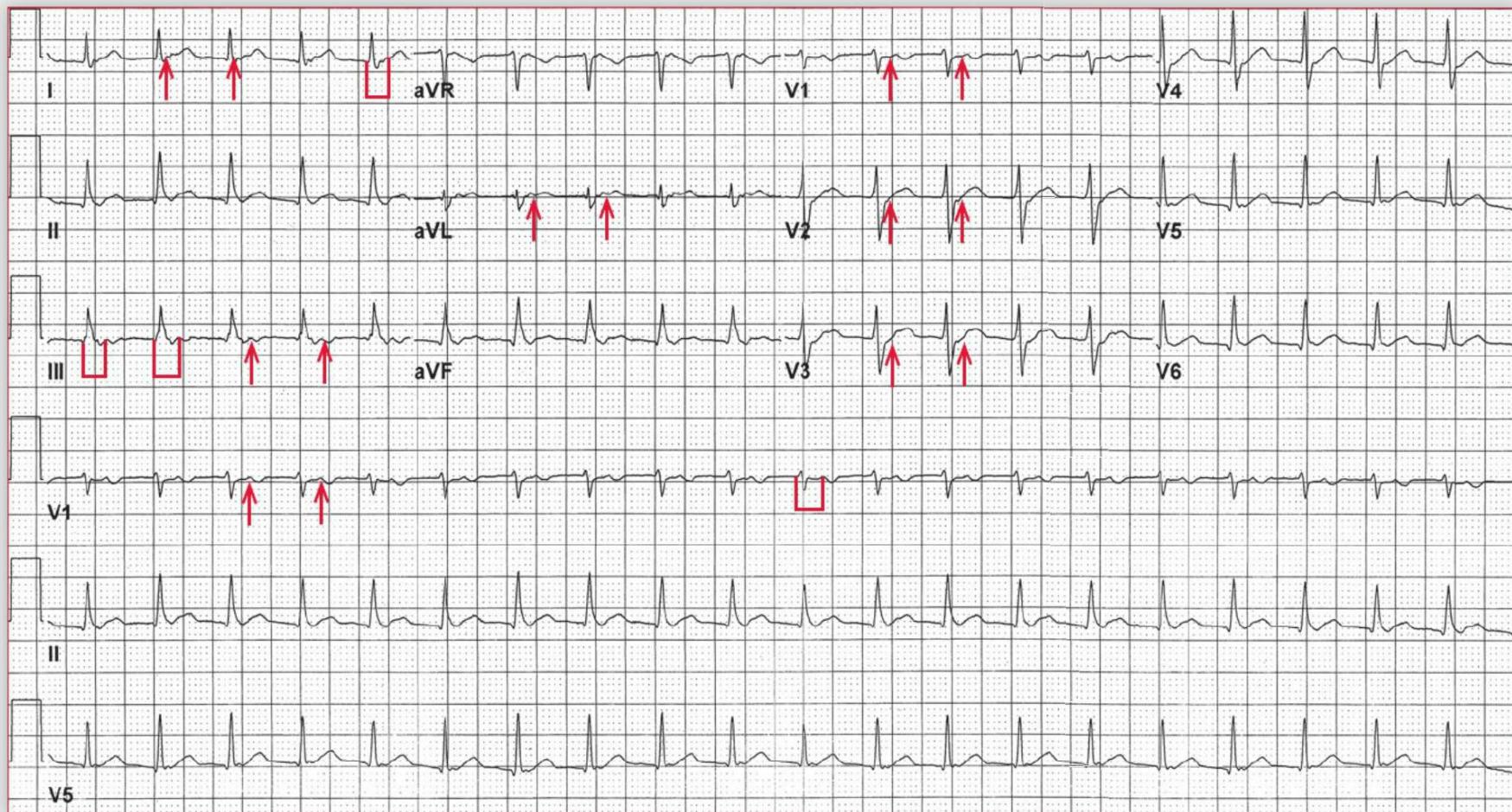


What abnormalities are evident on the initial ECG (35A)?

After treatment, what further information in the second tracing (ECG 35B) helps you make the diagnosis?

What further workup is indicated?

Based on the diagnosis and likely underlying pathology suggested by the history, what therapy is indicated?



ECG 35A Analysis: Short RP tachycardia, ectopic junctional tachycardia

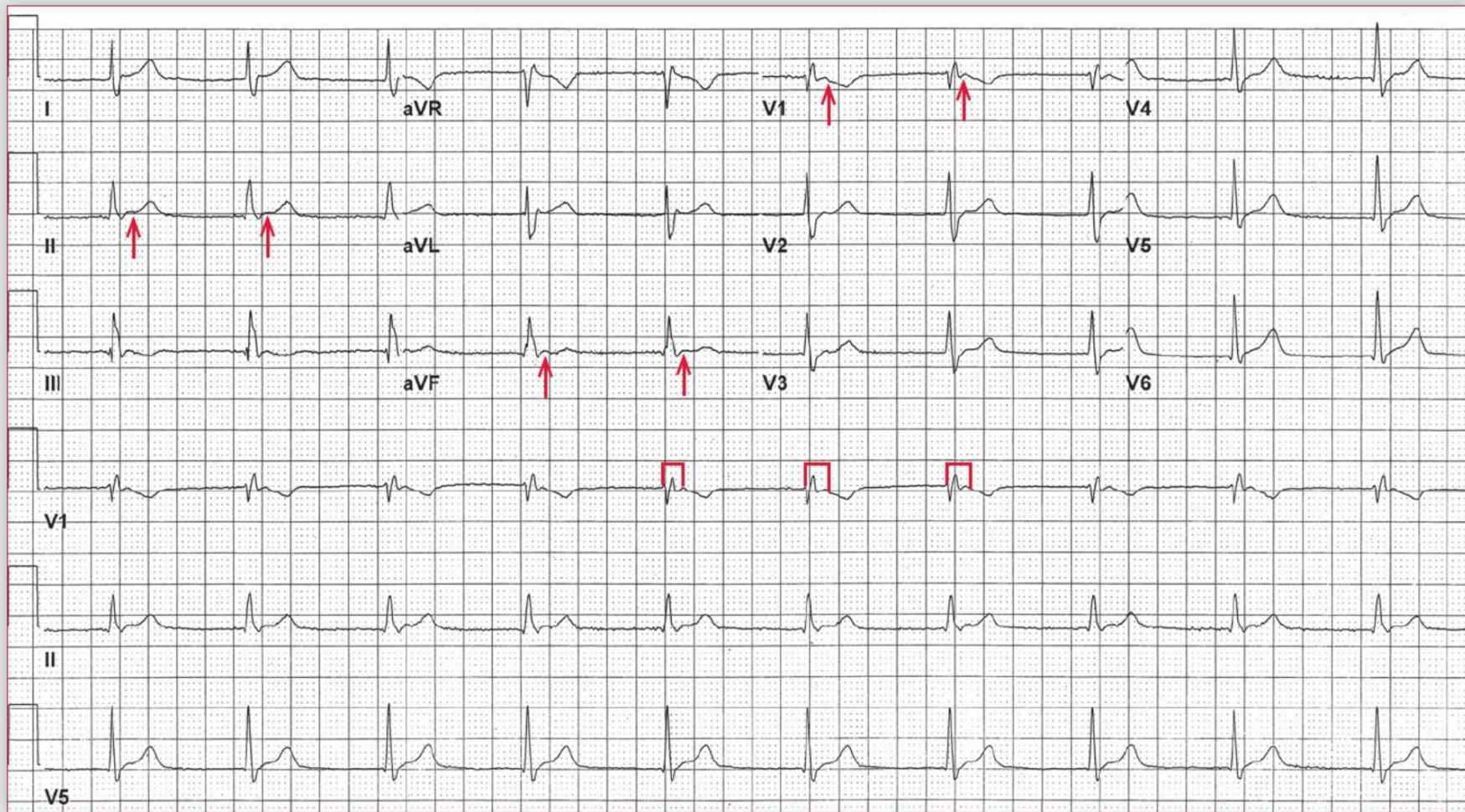
ECG 35A shows a regular rhythm at a rate of 130 bpm. The QRS complexes are of normal duration (0.08 sec) and morphology. The QT/QTc intervals are normal (300/400 msec). The axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). No P waves are seen before any of the QRS complexes. However, there appears to be a P wave at the end of the QRS complex, within the ST segment (↑). This is best seen in leads I, III, aVL, and V1. The RP interval (◻) is short (0.16 sec) but constant.

This is termed short RP tachycardia, for which there are a number of potential etiologies, including:

- Sinus tachycardia with a long PR interval
- Atrial flutter with 2:1 AV block (with a second flutter wave that is not obvious)
- Ectopic junctional tachycardia (and a retrograde P wave)
- Atrial tachycardia
- AV reentrant tachycardia (AVRT), as occurs with a preexcitation syndrome

• Typical (slow-fast) AV nodal reentrant tachycardia (AVNRT). Short RP tachycardia is an uncommon variant of typical AVNRT and has often been called slow-slow. The usual mechanism for typical AVNRT is dual AV nodal pathways (a slow pathway that conducts slowly but recovers quickly due to a short refractory period, and a fast pathway that conducts rapidly but recovers slowly due to a long refractory period). With typical AVNRT, antegrade conduction to the ventricles is via the slow pathway and retrograde conduction to the atria occurs via the fast pathway (slow-fast). There is simultaneous activation of the atria retrogradely and ventricles antegradely. As a result, the P wave and QRS complex are superimposed and hence no P wave is seen before or after the QRS complex (no RP tachycardia). However, if the fast pathway conduction is relatively slow, the circuit is called slow-slow and in this situation there is a slight delay in atrial activation, resulting in a short RP interval.

continues



ECG 35B Analysis: Ectopic junctional rhythm

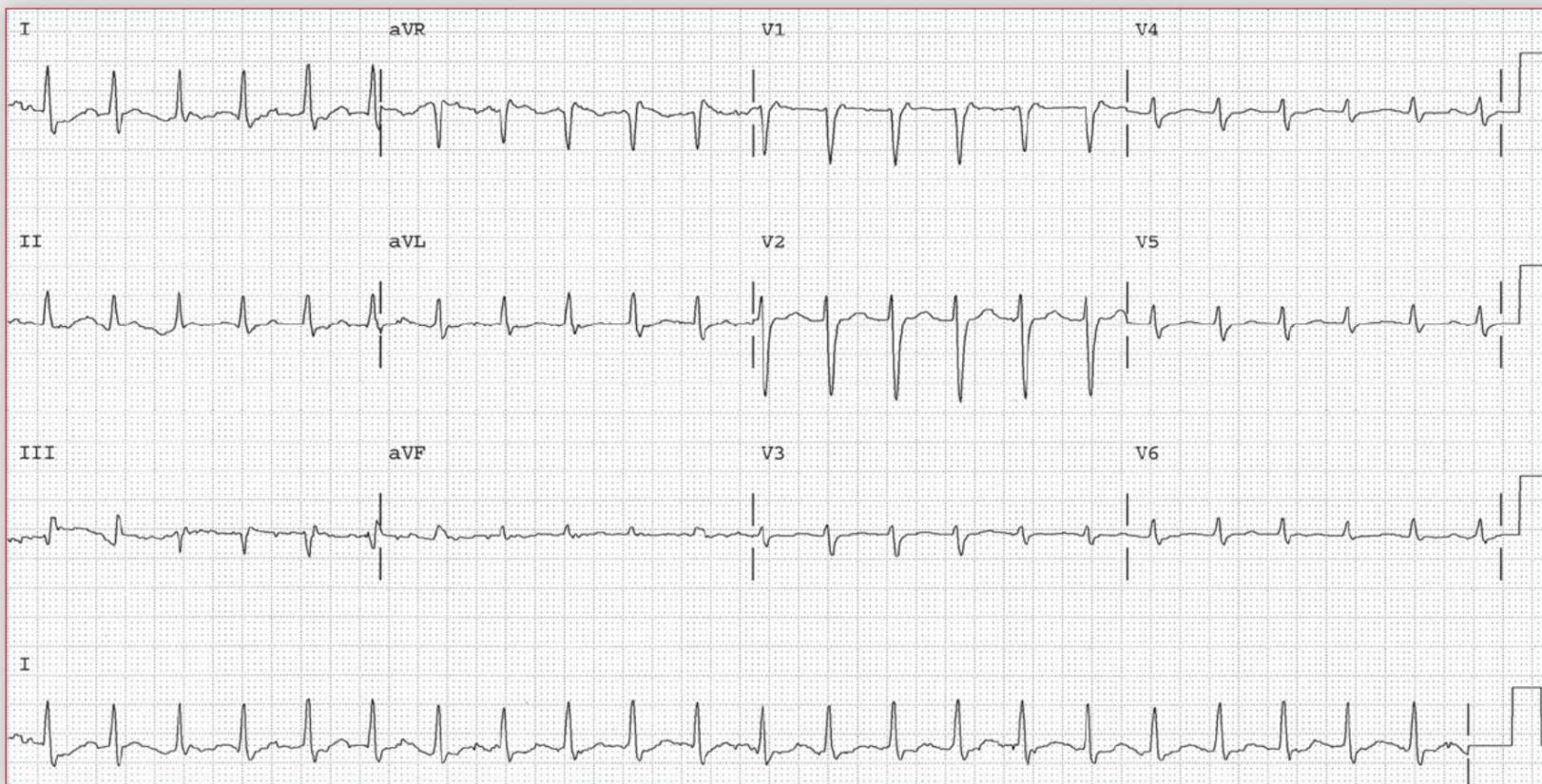
In ECG 35B, the rhythm is regular at a rate of 62 bpm. The QRS morphology, duration, and axis are normal and are similar to what is seen in ECG 35A. The QT/QTc intervals are normal (400/410 msec). There are no P waves before any QRS complex, but there is evidence of atrial activity after the QRS complex, particularly in leads II, aVF, and V1 (\uparrow). The RP interval is constant (\sqcap) and the same as that seen in ECG 35A (*ie*, 0.16 sec). This is, therefore, an ectopic junctional rhythm. The rhythm is called ectopic junctional tachycardia when the rate exceeds 100 bpm as a result of sympathetic stimulation. Hence the rhythm in ECG 35A is ectopic junctional tachycardia; the QRS morphology, presence of retrograde P wave, and RP interval are the same in ECG 35A and ECG 35B, the only difference being the rate.

The patient displays classic signs of acute thyrotoxicosis. Supraventricular tachyarrhythmias, especially atrial fibrillation, are a known manifestation of this disease. Evaluation of serum thyroid-stimulating hormone will establish the diagnosis. Intravenous followed by oral β -blockers are appropriate treatment for both the tachycardia and control of thyrotoxicosis symptoms. β -blockers are the drug of choice for therapy of arrhythmias due to thyrotoxicosis because the arrhythmias are the result of an increased sympathetic state. Digoxin may be added for control of refractory tachycardias; however, the digoxin doses required to control hyperthyroid-associated arrhythmias are usually quite high. Acetaminophen or aspirin should be used to control the temperature. Administration of a thionamide will suppress thyroid hormone production. Further therapy will depend on the underlying cause. ■

Core Case 36

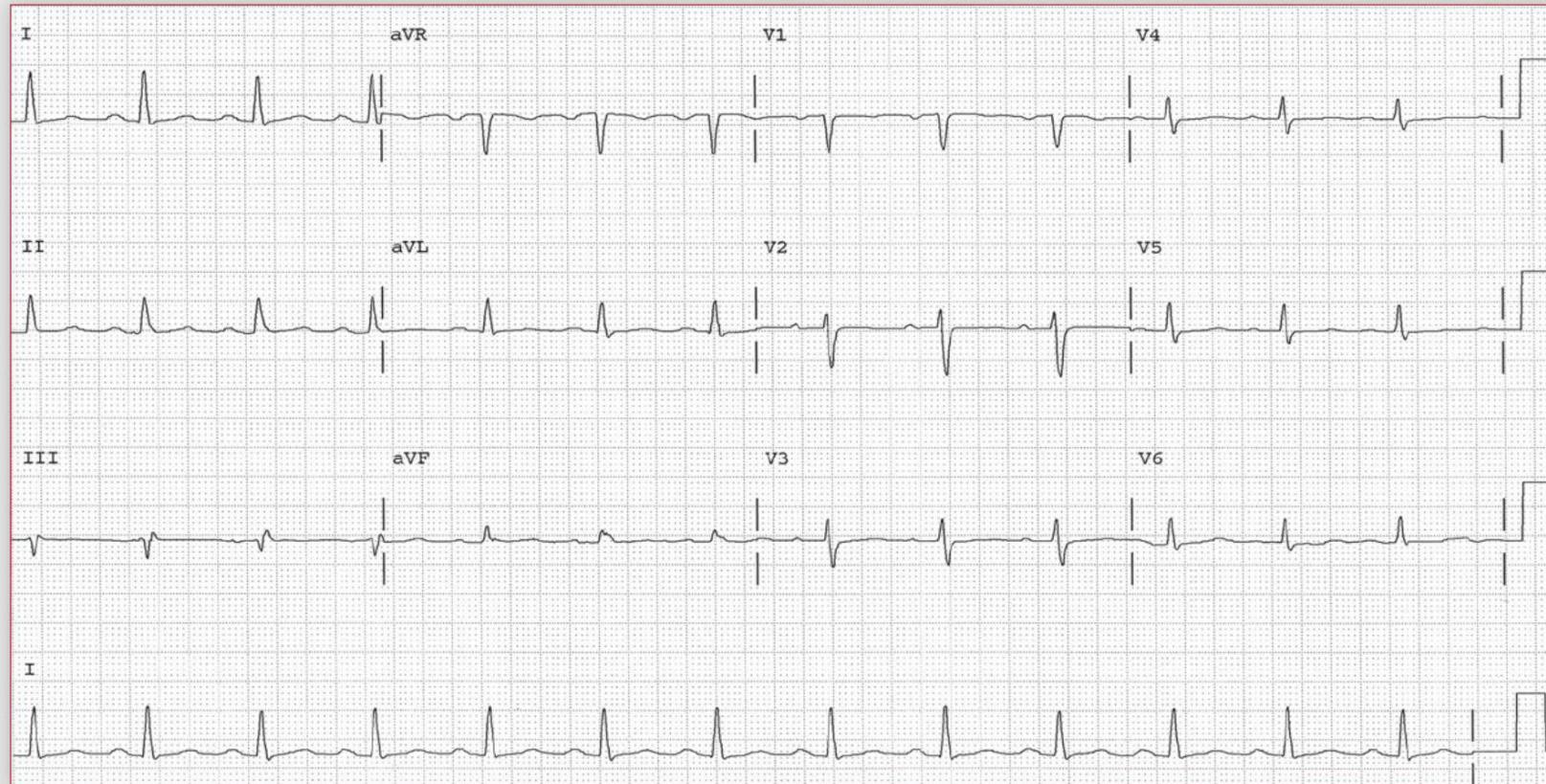
An 18-year-old man presents to the emergency department with 1 day of “off-and-on fluttering” in his chest. The sensation initiates and terminates without warning, and lasts several minutes to an hour. He states that he may have had similar short-lived episodes in the past, but this is prolonged and worrisome, prompting his presentation. He denies lightheadedness.

ECG 36A



As he is placed on telemetric monitoring, his heart rate is noted to be 140 bpm. He is complaining of his usual symptom of fluttering. An ECG is obtained (ECG 36A). Several minutes later, without intervention, his heart rate abruptly slows to 78 bpm. A second ECG is obtained (ECG 36B).

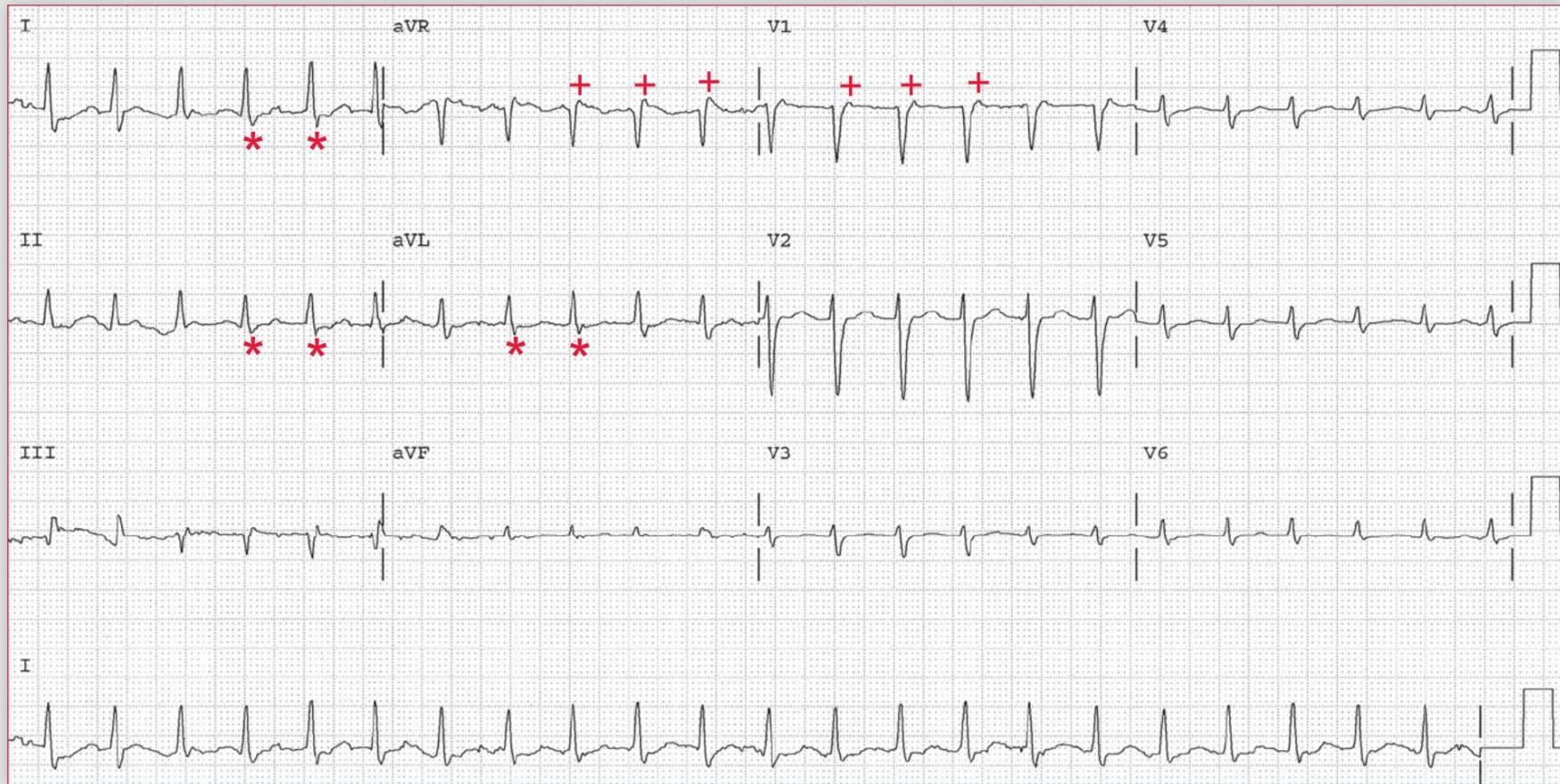
ECG 36B



What abnormalities are notable in ECG 36A?

What is the cause of the patient's symptoms?

Comparing ECGs 36A and 36B, what is the patient's diagnosis and what is the pathophysiology of this condition?



ECG 36A Analysis: No RP tachycardia (typical AV nodal reentrant tachycardia [AVNRT]), clockwise rotation (poor R-wave progression and late transition)

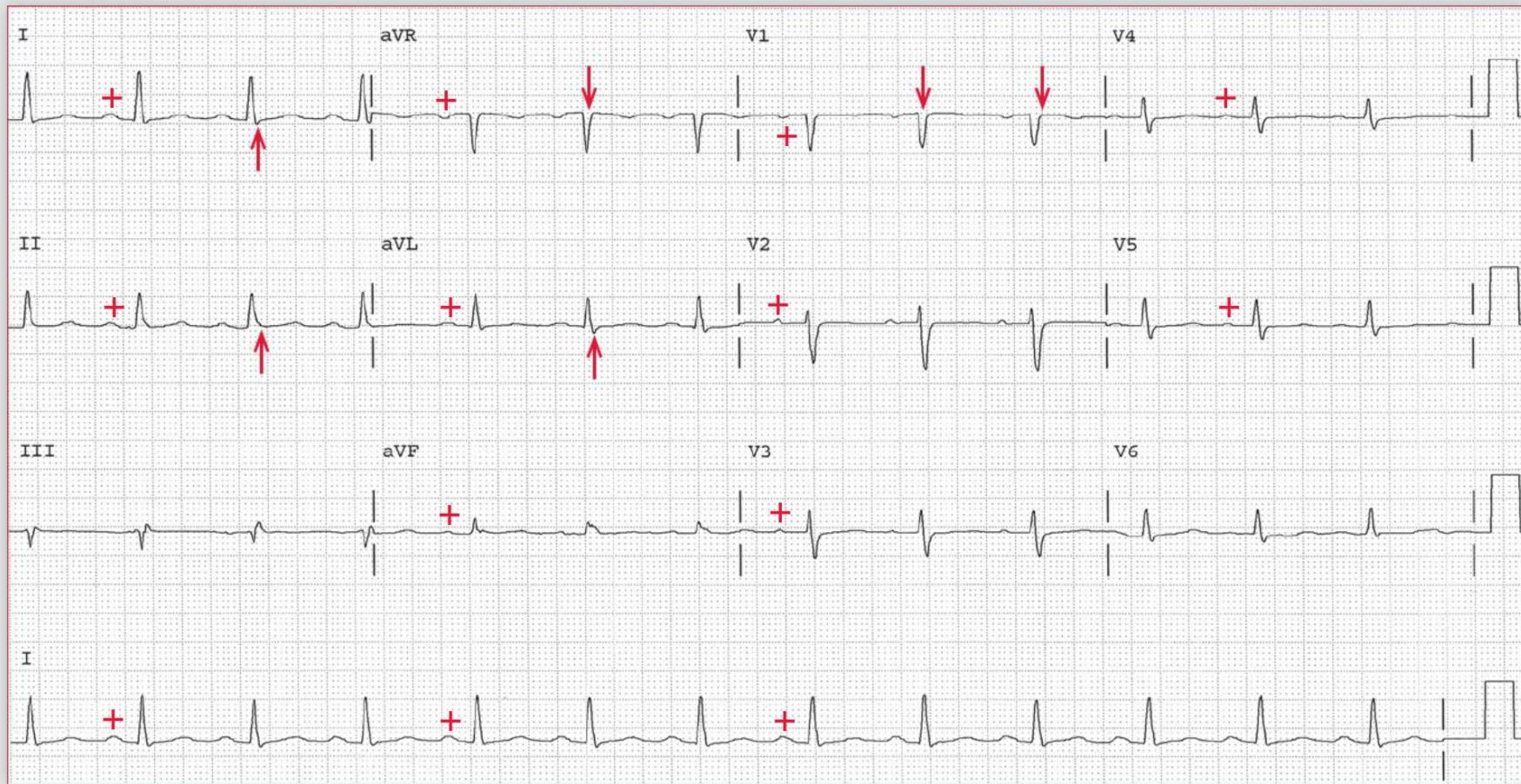
ECG 36A shows a regular rhythm at a rate of 140 bpm. The QRS complex has a normal duration (0.08 sec) and morphology. The QT/QTc intervals are normal (300/450 msec). The axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There are no obvious P waves before or after the QRS complex. This is, therefore, no RP tachycardia. There is a slight abnormality superimposed on the terminal portion of the QRS complex, particularly in leads aVR and V1, that resembles an R' waveform (+). There are also small S waves in leads I, II, and aVL (*). Although these waveforms do not look like P waves, they are suggestive of atrial activity. Comparison with a baseline ECG in sinus rhythm would be useful.

Since there are no obvious P waves before or after the QRS complex, the most likely etiology for this regular narrow complex supraventricular tachycardia is typical AV nodal reentrant tachycardia (AVNRT) (*ie*, slow-fast). It is identified by a regular supraventricular tachycardia at a rate between 140 and 220 bpm without any clear P waves before or after the QRS complex. The P wave (which is retrograde) may be superimposed on the end of the QRS complex, appearing to be an R' waveform or resembling an S wave.

AVNRT is a form of a junctional tachycardia. However, in contrast to ectopic junctional tachycardia, it is not the result of an ectopic focus in the AV node or junction. The mechanism for AVNRT is reentry resulting from dual AV nodal pathways. One pathway conducts slowly (slow pathway) but has a short refractory period (recovers more quickly),

while the other pathway conducts rapidly (fast pathway) but has a long refractory period (recovers more slowly). These dual pathways form a circuit, connected proximally by the atrial myocardium and distally within the bundle of His. During sinus rhythm the impulse is conducted to the ventricle via the fast pathway, while the slow pathway is inactive. If there is a premature atrial complex that is appropriately timed such that the fast pathway has not yet recovered, this premature impulse is conducted antegradely to the ventricles via the slow pathway, which recovers more quickly, with a long PR interval as it conducts slowly. If the impulse reaches the distal end of the fast pathway after it has recovered, it can penetrate the fast pathway in a retrograde direction and conduct to the atria to activate them retrogradely. At the same time there is antegrade activation of the ventricles. If the retrograde impulse reaches the slow pathway when it has recovered, it may reenter the slow pathway and again conduct antegradely. If this process continues (antegrade through the slow pathway and retrograde through the fast pathway), AVNRT is generated. Since retrograde conduction to the atria is by a fast conducting pathway, atrial activation occurs simultaneously or almost simultaneously with antegrade ventricular activation. Hence there is no obvious P wave seen, as it occurs during the QRS complex or may be superimposed at the very end of the QRS complex, with an R' morphology (particularly in lead V1) or what appears to be a terminal S wave (particularly in the limb leads or leads V5-V6). This is called slow-fast AVNRT and is the mechanism for typical AVNRT.

continues



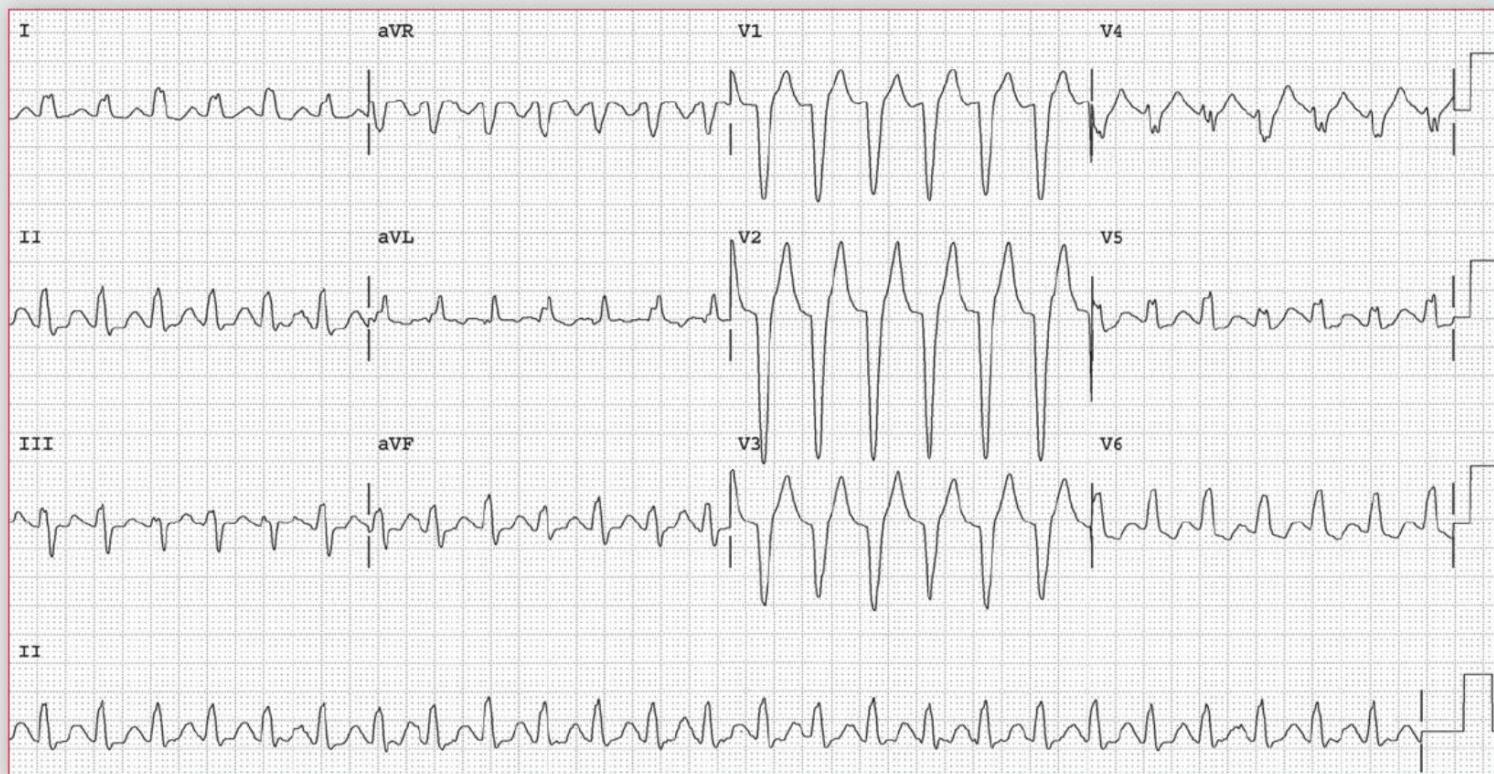
ECG 36B Analysis: Normal sinus rhythm, first-degree AV block
(prolonged AV conduction)

ECG 36B shows a regular rhythm at a rate of 78 bpm. The QRS complex duration, morphology, and axis are the same as in ECG 36A. The QT/QTc intervals are normal (390/440 msec). There are P waves (+) in front of each QRS complex with a stable PR interval (0.24 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with first-degree AV block or prolonged AV conduction. Noted is that the R' waveform in leads aVR and V1 (↓) and the terminal S wave in leads I, II, and aVL (↑) are not present during sinus rhythm, confirming the fact that this was indeed a retrograde P wave. ■

Core Case 37

A 45-year-old man with a prior myocardial infarction presents to his primary care physician on an urgent basis. He has had several episodes of “racing heart” over the course of the morning and a feeling like his heart was coming out of his chest. These episodes have lasted several minutes at a time, and he has felt lightheaded during a few of them. He denies chest pain but admits to concurrent dyspnea. On further questioning, he states he has had similar episodes in the past, but they have been rare, isolated, and short lived, and he did

ECG 37A



not seek medical attention for them. He was having symptoms at the time he was being seen.

On exam, he appears in mild distress. He is diaphoretic with a rapid radial pulse. His lungs are clear. His jugular venous pressure is not elevated. His apical impulse is not displaced. Heaves are absent. S1 and S2 sound regular in cadence, and gallops and murmurs are not appreciated.

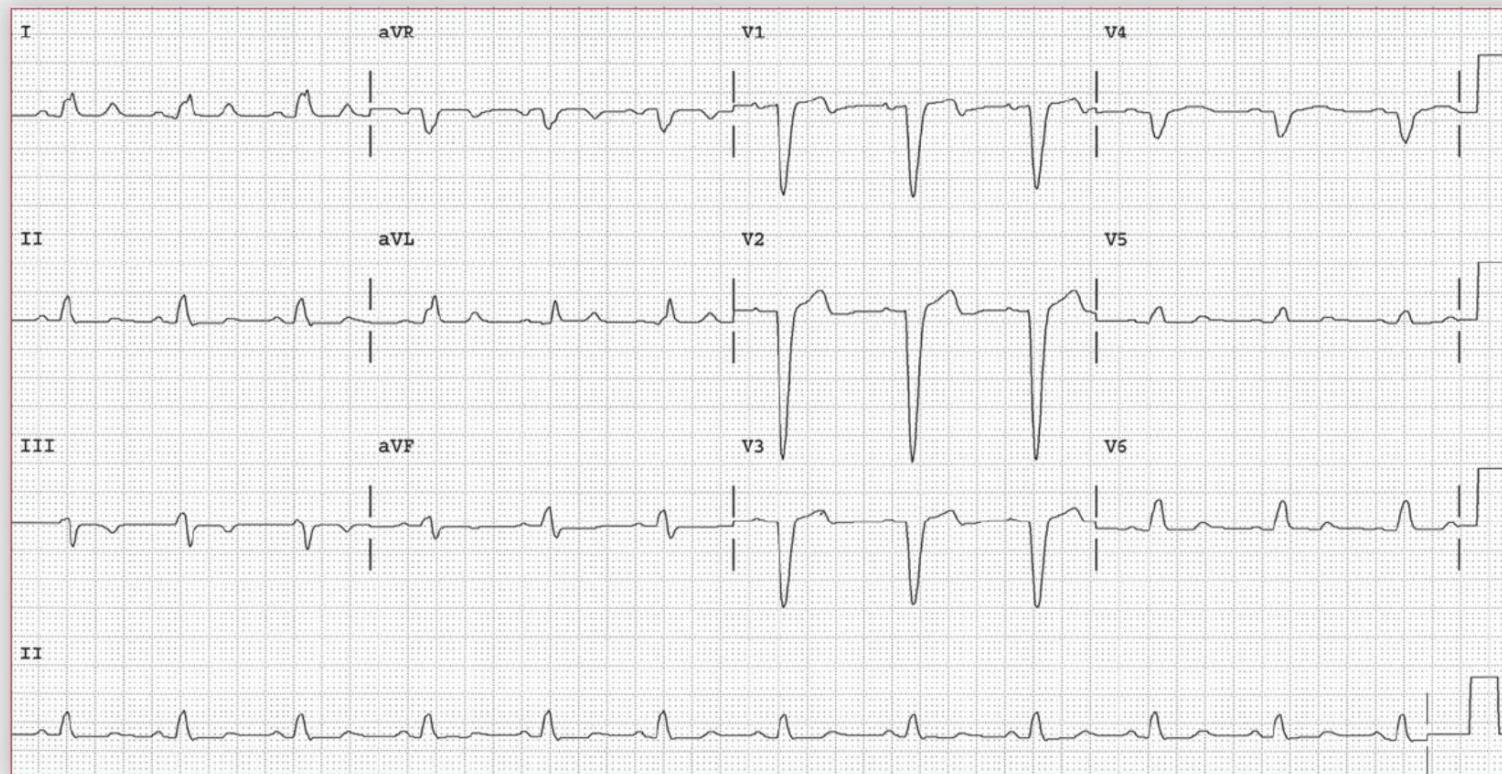
An ECG is obtained (ECG 37A). Suddenly, he states that the sensation has passed. A second ECG is obtained (ECG 37B)

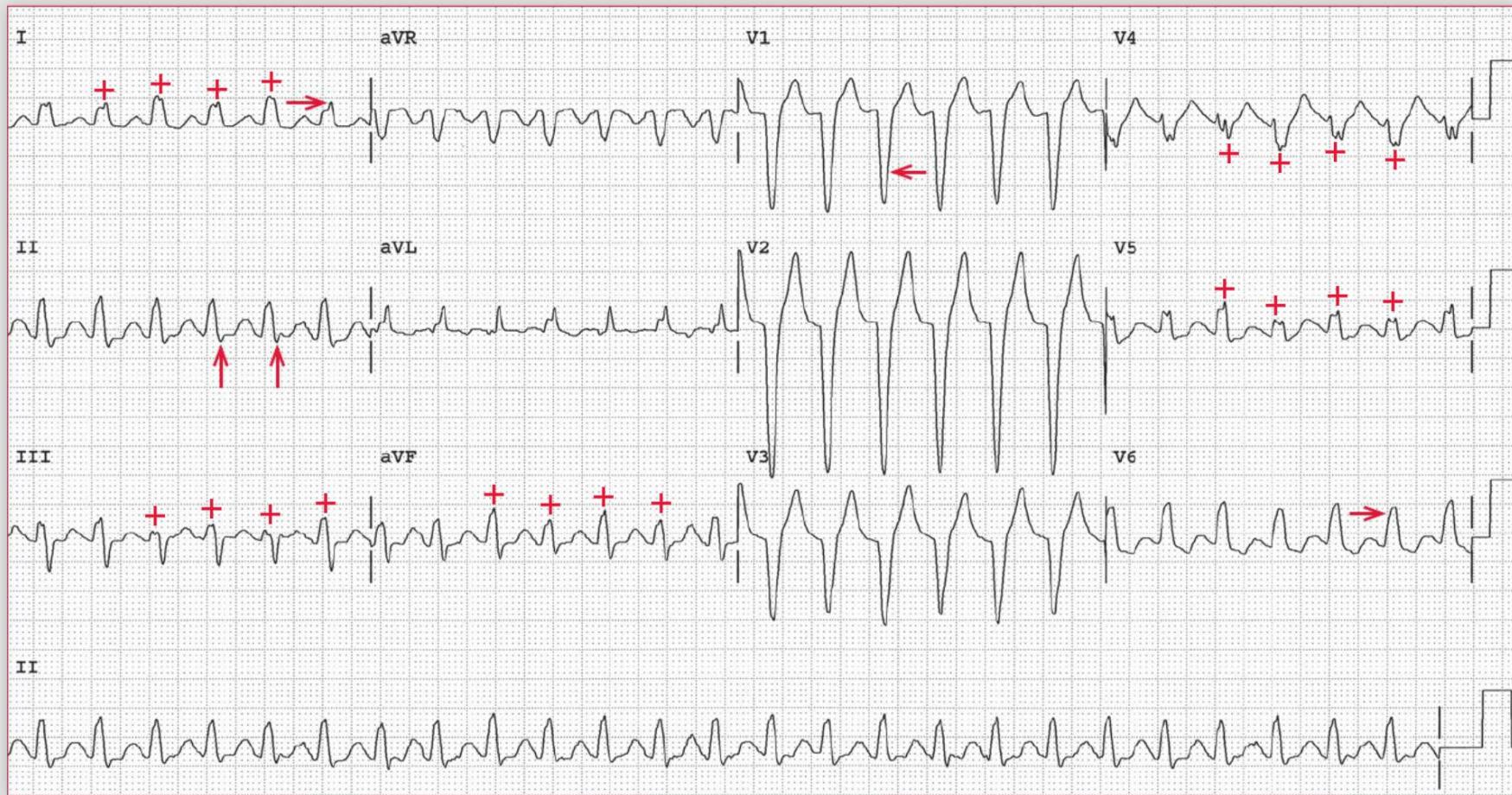
What abnormalities are notable in ECG 37A?

What is the differential diagnosis?

When comparing the two ECGs, what diagnosis is strongly suggested?

ECG 37B





ECG 37A Analysis: No RP tachycardia (AV nodal reentrant tachycardia [AVNRT]), left bundle branch block (LBBB), QRS or electrical alternans

ECG 37A shows a regular rhythm at a rate of 154 bpm. The QRS complex duration is increased (0.14 sec). There is a broad R wave in leads I and V5-V6 (→) and a deep QS complex in lead V1 (←), characteristic of typical left bundle branch block (LBBB). The QT/QTc intervals are prolonged (300/480 msec) but are normal when corrected for the prolonged QRS complex duration (240/380 msec). No P waves are seen before or after any of the QRS complexes. Hence this is termed no RP tachycardia. The etiology may be either ventricular tachycardia or supraventricular (junctional) tachycardia with aberration. The most common etiology for supraventricular or junctional tachycardia that is not associated with any obvious P waves is AV nodal reentrant tachycardia (AVNRT). In this case it is associated with an LBBB, which is either rate related or preexisting, being present during normal sinus rhythm. The presence of a QRS complex with a typical LBBB morphology strongly suggests that the arrhythmia is supraventricular.

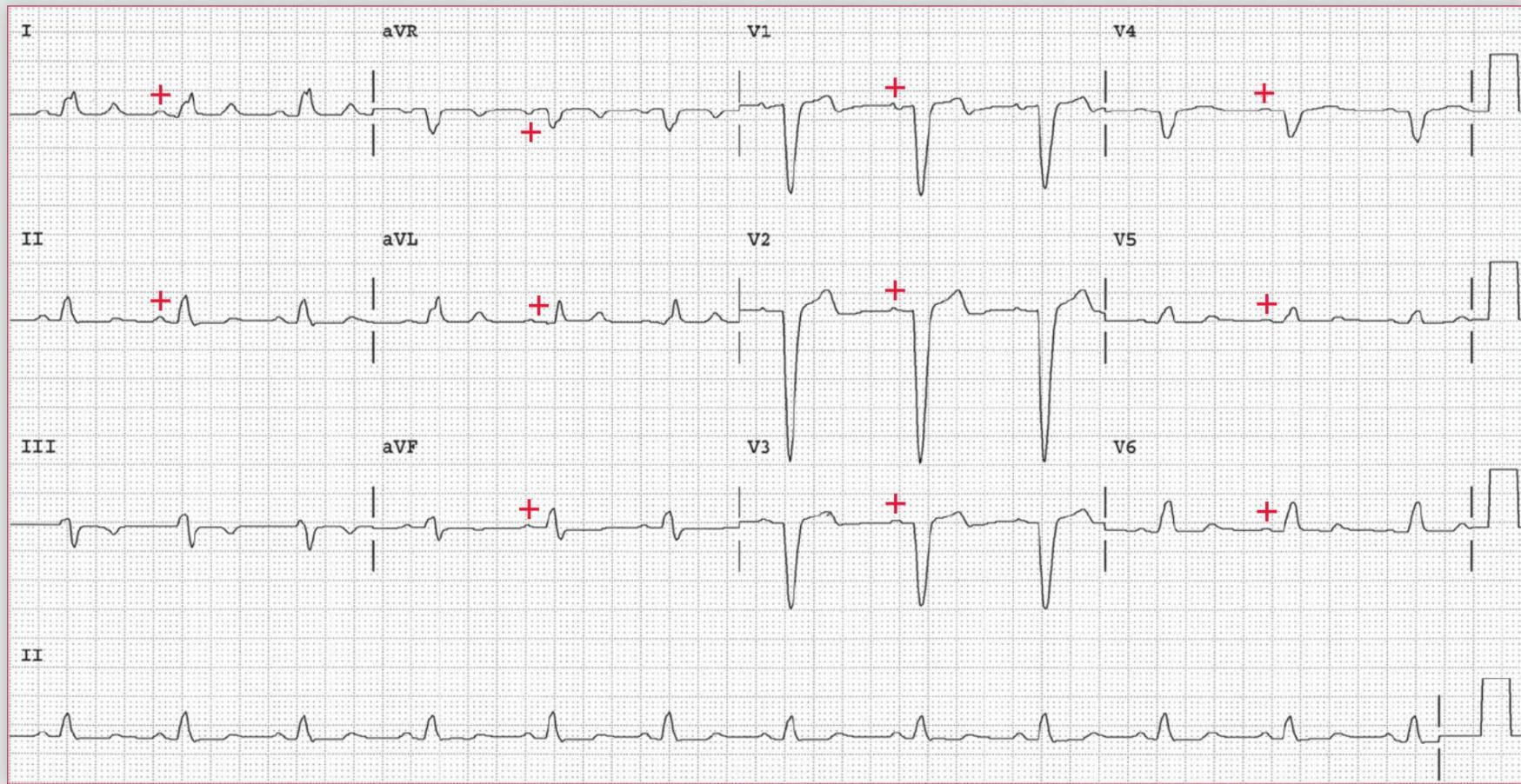
Although P waves are not seen, there is a small terminal S wave in lead II (↑) that is suggestive of a P wave. To establish this as a retrograde P wave, the morphology of the QRS complex during the tachycardia should be compared with the QRS complex morphology during sinus rhythm.

In addition, there is evidence of beat-to-beat changes in QRS complex amplitude (+), termed QRS (electrical) alternans, which is most prominent in leads I, III, aVF, V4, and V5. Although most often considered a hallmark of cardiac tamponade, QRS alternans may be seen in any rapid supraventricular tachycardia. It is not generally seen with ventricular tachycardia. Alterations in cardiomyocyte cytosolic calcium concentrations provoked by rapid cellular depolarization-repolarization cycles of elevated heart rates can result in a subtle change in the electrical axis of the heart and the QRS complex amplitude. Often also seen is T-wave alternans. During supraventricular tachycardias, QRS alternans is evident in ventricular but not atrial cells; therefore, electrical alternans is limited to the QRS complex. P-wave alternans is not seen in supraventricular tachycardia, but it can be seen in cardiac tamponade when the heart itself is swinging in a fluid-filled pericardium. In this situation there is beat-to-beat alteration in all the ECG waveforms (QRS complex, T wave, and P wave).

Other causes of electrical or QRS alternans, also the result of alterations in calcium fluxes, include acute myocardial infarction, decompensated heart failure, and severe cardiomyopathy.

continues

Podrid's Real-World ECGs



ECG 37B Analysis: Normal sinus rhythm, LBBB

In ECG 37B the rhythm is regular at a rate of 70 bpm. The QRS complex has an LBBB morphology and is identical to the QRS morphology seen in ECG 37A. This confirms that the tachycardia in ECG 37A is supraventricular. The QT/QTc intervals are normal (420/450 msec; 360/390 msec when corrected for the prolonged QRS complex duration). There is a P wave (+) before each QRS complex, and the PR interval is constant (0.18 sec). The P wave is positive in leads I, II,

aVF, and V4-V6. This is a normal sinus rhythm. Since there is an LBBB present during the baseline normal sinus rhythm, the wide complex tachycardia in ECG 37A, which also has an LBBB, is supraventricular with an LBBB that is present at baseline; hence this is AVNRT with an underlying LBBB. Also noted is the fact that the small S wave in lead II during tachycardia is not present with sinus rhythm, confirming that it was indeed the retrograde P wave. ■

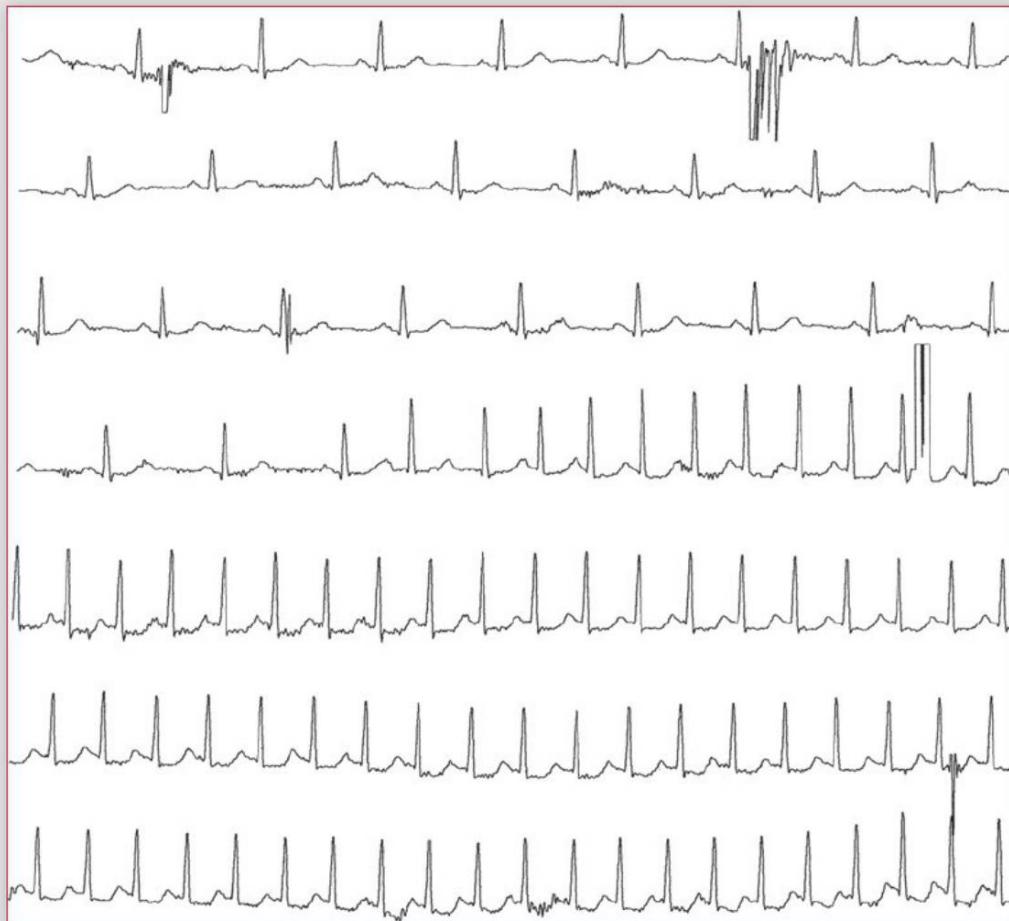
Notes

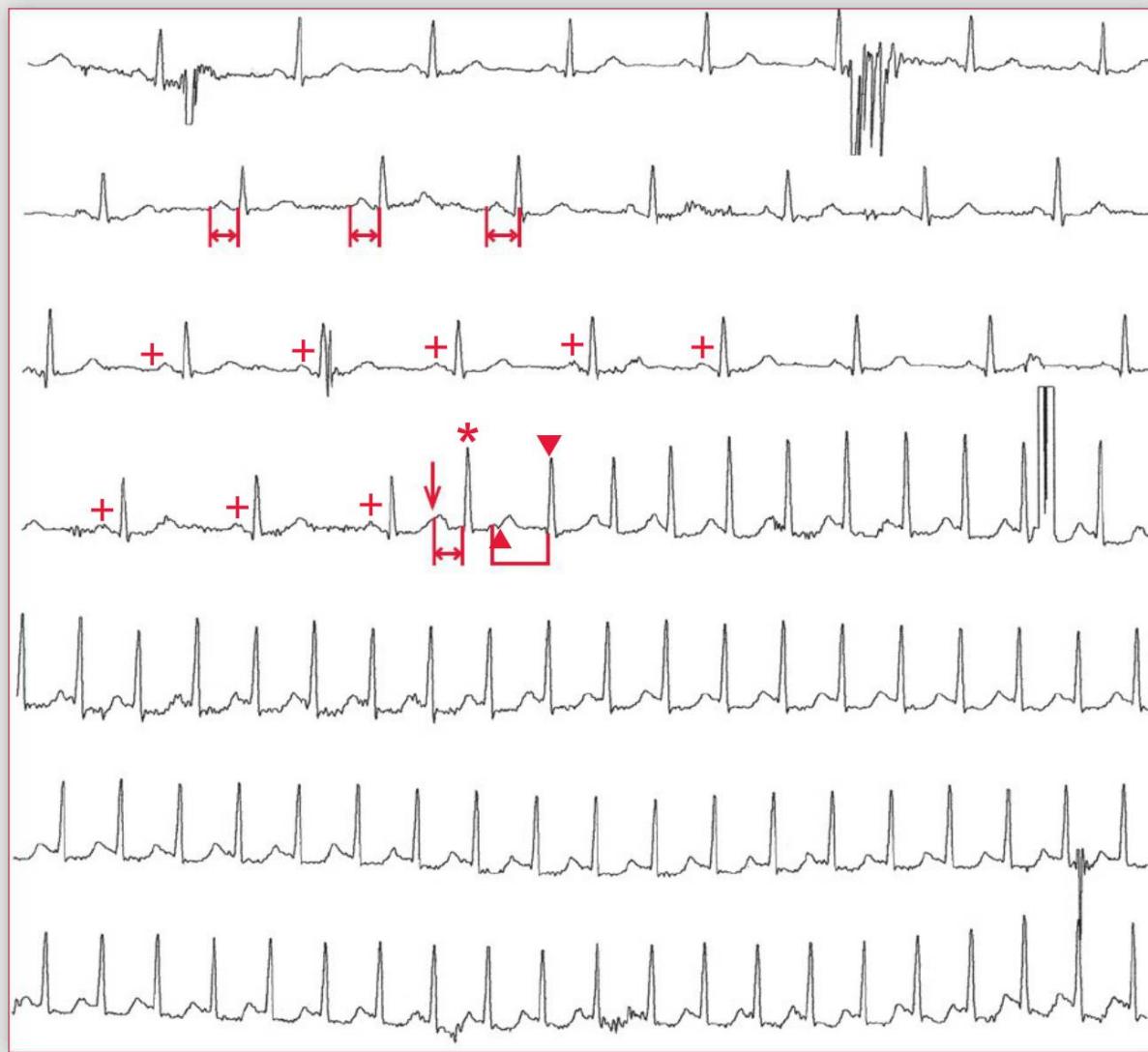
An 18-year-old man is brought to the emergency department after being arrested for disorderly conduct. According to his friend, the man ingested an unknown illicit drug in pill form. Shortly afterward, he became agitated and belligerent.

In the emergency department the patient is restrained both chemically and physically. As part of his workup, telemetric monitoring is set up. The nurse notices a sudden increase in his heart rate. You review the tracing just prior to her observation.

What rhythm abnormality can be seen on the ECG?

What is the likely electrocardiographic diagnosis?





ECG 38 Analysis: Sinus rhythm, premature atrial complex, AV nodal reentrant tachycardia

The rhythm strip initially shows a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex with a PR interval (↔) that is constant (0.16 sec). The fourth QRS complex (*) on the fourth line is early and is preceded by an early P wave (↓) that is superimposed on the T wave, altering its morphology. The PR interval (↔) of this premature atrial complex is the same as the PR interval of the sinus complex. The fifth QRS complex is also premature (▼) and also preceded by a P wave (▲); this is the waveform that causes a notching of the ST segment. The PR interval prior to the fifth QRS complex is very long (□) (0.38 sec). This is a second premature atrial complex after which there is supraventricular tachycardia at a rate of 168 bpm. The QRS complex of the tachycardia is the same as the sinus complex. There are no P waves before or after the QRS complexes of the tachycardia. This is, therefore, a no RP tachycardia, the most common etiology for which is AV nodal reentrant tachycardia (AVNRT) that is initiated by a premature atrial complex with a very long PR interval.

The mechanism for AVNRT is reentry resulting from dual AV nodal pathways. One pathway conducts slowly (slow pathway) but has a short refractory period (recovers more quickly), while the other pathway conducts rapidly (fast pathway) but has a long refractory period (recovers more slowly). These dual pathways form a circuit, connected

proximally by the atrial myocardium and distally within the bundle of His. During sinus rhythm the impulse is conducted to the ventricle via the fast pathway, while the slow pathway is inactive. If there is a premature atrial complex that is appropriately timed such that the fast pathway has not yet recovered, this premature impulse is conducted antegradely to the ventricle via the slow pathway, which recovers more quickly, with a long PR interval as it conducts slowly. If the impulse reaches the distal end of the fast pathway after it has recovered, it can penetrate and conduct via the fast pathway in a retrograde direction and activate the atria retrogradely. At the same time there is antegrade activation of the ventricles. If the retrograde impulse reaches the slow pathway when it has recovered, it may reenter the slow pathway and again conduct antegradely. If this process continues (antegrade through the slow pathway and retrograde through the fast pathway), AVNRT is generated. Since conduction to the atria is by a fast conducting pathway, atrial activation occurs simultaneously or almost simultaneously with antegrade ventricular activation. In this situation no obvious P wave is seen, as it occurs during the QRS complex or may be superimposed at the very end of the QRS complex, resembling an R' morphology (particularly in lead V1) or terminal S wave. This is called slow-fast AVNRT and is the mechanism for typical AVNRT. ■

Core Case 39

An 18-year-old man is seen by his primary care physician for a complaint of palpitations. He was recently seen in the emergency department

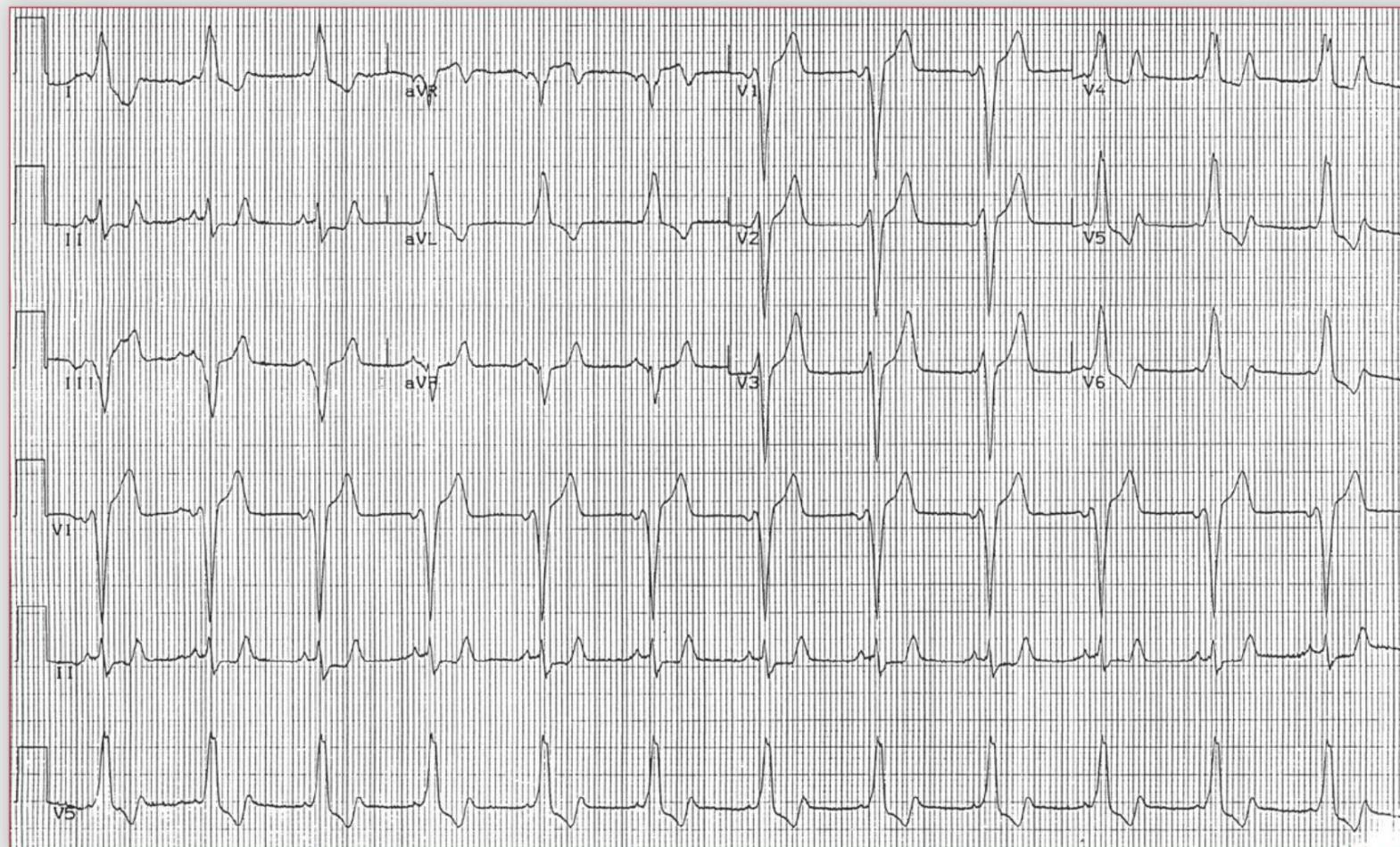
ECG 39A



during one of these episodes and brings a copy of his ECG to the office (ECG 39A). The ECG obtained during this office visit (ECG 39B) is also shown.

**What does ECG 39A show?
Considering both ECGs, what is the diagnosis?
What acute treatment is appropriate?**

ECG 39B





ECG 39A Analysis: Short RP tachycardia, orthodromic AV reentrant tachycardia

The rhythm in ECG 39A is regular at a rate of 180 bpm. The QRS complex has a normal duration (0.08 sec), morphology, and axis in the frontal plane, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). There is minimal J-point and ST-segment depression seen in leads V3-V5 (\uparrow). The ST-segment depression is upsloping. The QT/QTc intervals are normal (260/450 msec).

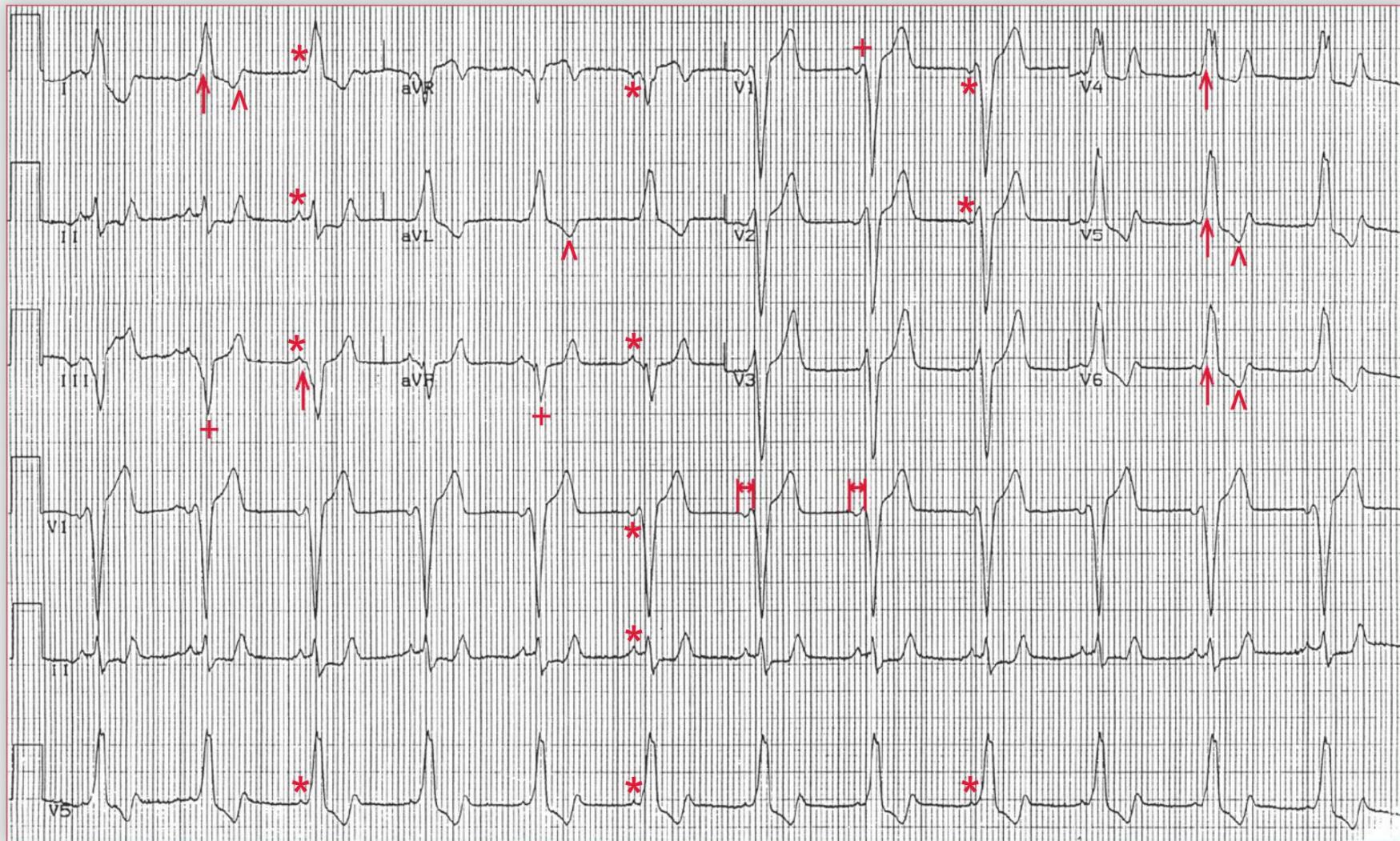
No obvious P waves are seen before any QRS complex. However, a small notching or waveform can be seen after each QRS complex on the T wave, especially in leads III and V1 (\downarrow). T waves should have a smooth upstroke and downstroke. Notching of the T wave suggests a superimposed P wave. Therefore, this waveform represents the P wave.

Hence this is a short RP tachycardia. There are a number of possible explanations for a short RP tachycardia:

- Sinus tachycardia with a long PR interval
- Atrial flutter with 2:1 AV block (with one of the flutter waves not obvious)
- Ectopic junctional tachycardia
- Atrial tachycardia
- AV reentrant tachycardia (AVRT)
- Typical (slow-fast) AV nodal reentrant tachycardia (AVNRT), but an unusual variant termed slow-slow

Based on this ECG, this is short RP tachycardia, but the actual etiology cannot be established.

continues



ECG 39B Analysis: Sinus rhythm, Wolff-Parkinson-White pattern

ECG 39B, which was obtained in the office while the patient was asymptomatic, is very helpful in this case. The rhythm is regular at a rate of 74 bpm. The QRS complex duration is increased (0.14 sec) and there is a prolonged and slurred upstroke to the QRS complex (\uparrow), especially evident in leads I, III, and V4-V6. This is a delta wave. The delta wave results from early impulse conduction through an accessory pathway that bypasses the AV node and produces early initial direct myocardial activation that is slow and abnormal. There is also impulse conduction through the normal AV node–His-Purkinje system. This impulse is delayed but will join with the earlier impulse that occurs via the accessory pathway. The result is a fusion complex that causes the base of the QRS complex to widen (as a result of the delta wave) while the peak of the QRS complex is narrower, resulting from impulse conduction through the normal His-Purkinje system. The QT/QTc intervals are prolonged (420/470 msec) but are normal when corrected for the prolonged QRS complex duration (360/400 msec). There is a P wave (*) before each QRS complex with a stable, although short, PR interval (0.10 sec) [\leftrightarrow]. The PR interval is short as a result of preexcitation or early myocardial activation via the accessory pathway. This is Wolff-Parkinson-White (WPW) pattern. The bypass is left posteroseptal as there is a pseudo inferior wall myocardial infarction pattern (Q waves in leads III and aVF and positive delta wave in lead V1 [+]). There are T-wave inversions (\wedge) in leads I, aVL, and V5-V6 that are repolarization abnormalities due to the abnormal ventricular activation via the accessory pathway. Importantly, abnormalities affecting the ventricles cannot be interpreted reliably in the presence of WPW pattern because ventricular activation is initially via the accessory

pathway and not the normal His-Purkinje system; this results in direct myocardial activation. It is for this reason that the infarction pattern is termed “pseudo,” because ventricular abnormalities cannot be diagnosed reliably when there is direct myocardial activation.

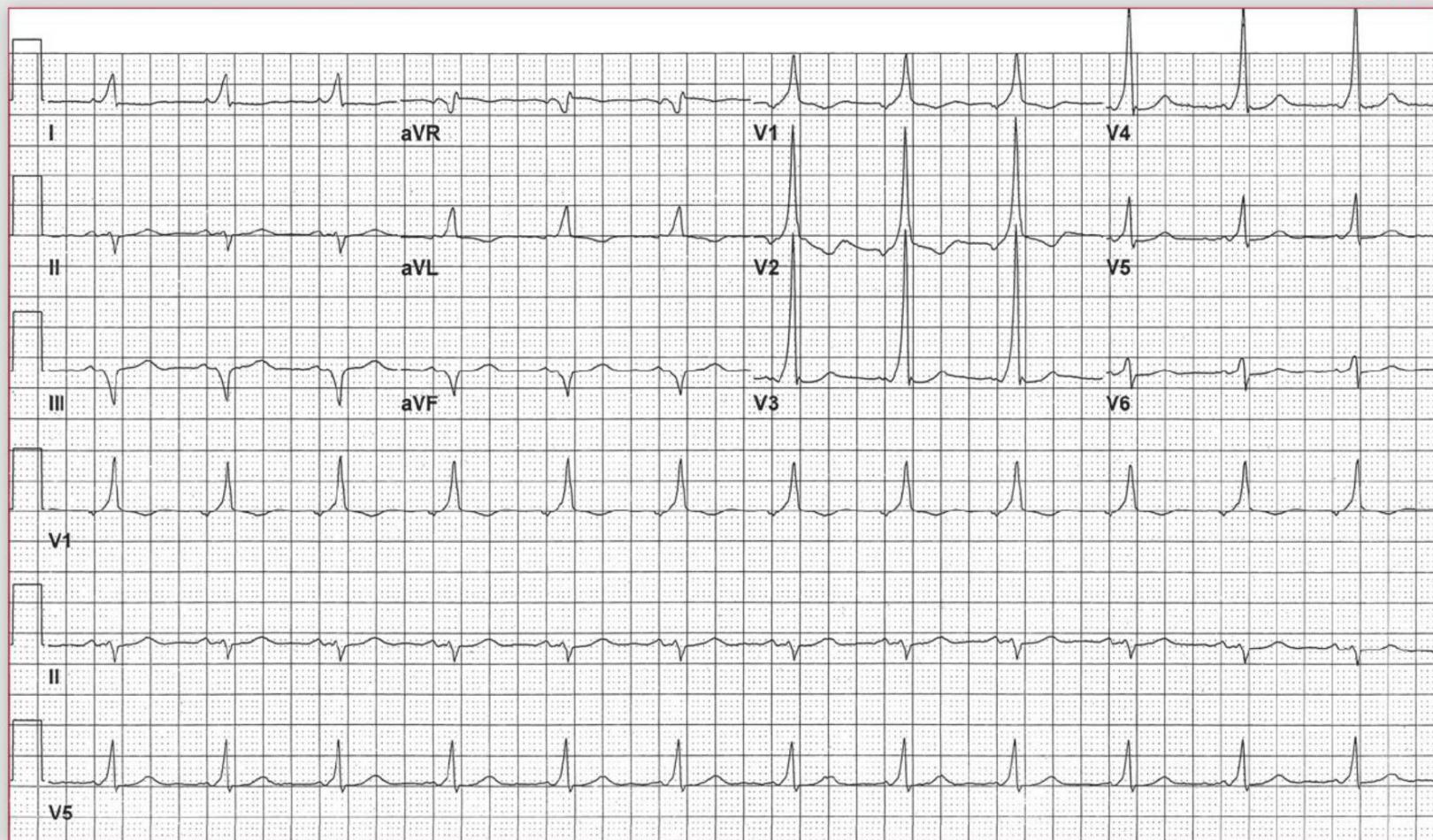
As there is a WPW pattern on the baseline ECG, the etiology for the short RP tachycardia in ECG 39A is orthodromic AVRT.

Orthodromic AVRT results from impulse conduction around a fixed circuit that involves the accessory pathway and the normal AV node–His-Purkinje pathway, which are linked proximally via the atrial myocardium and distally via the ventricular myocardium. This, therefore, is a macro-reentrant circuit. With orthodromic AVRT, the ventricles are activated by antegrade conduction via the normal AV node–His-Purkinje system with retrograde activation of the atria via the accessory pathway. As ventricular activation occurs via the normal His-Purkinje system, the QRS complex is narrow with a supraventricular morphology. However, a rate-related bundle branch block may occur, in which case the QRS complex will be wide with a typical bundle branch block morphology. Since the AV node is part of the circuit, any changes in AV nodal electrophysiologic parameters will terminate the arrhythmia. On occasion, enhancement of vagal effect on the AV node (as with carotid sinus pressure or Valsalva maneuver) will terminate the arrhythmia. Effective pharmacologic therapy for terminating the arrhythmia is adenosine, verapamil, diltiazem, β -blocker, or digoxin. If these agents are not effective, electrocardioversion can be used. ■

Core Case 40

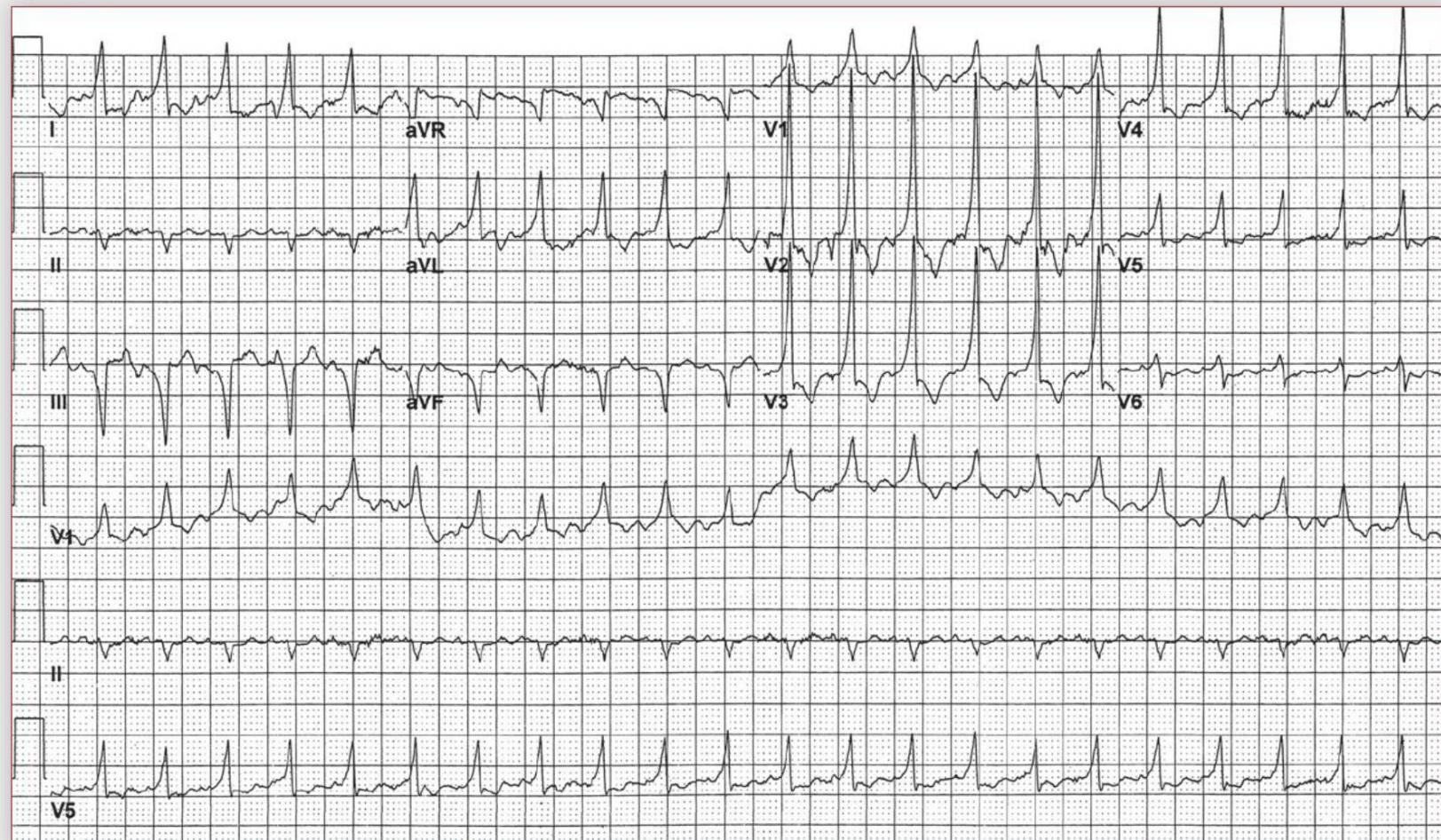
A 46-year-old woman with a history of infrequent palpitations comes to your office for evaluation. She has reported her symptoms to many prior health care providers

ECG 40A



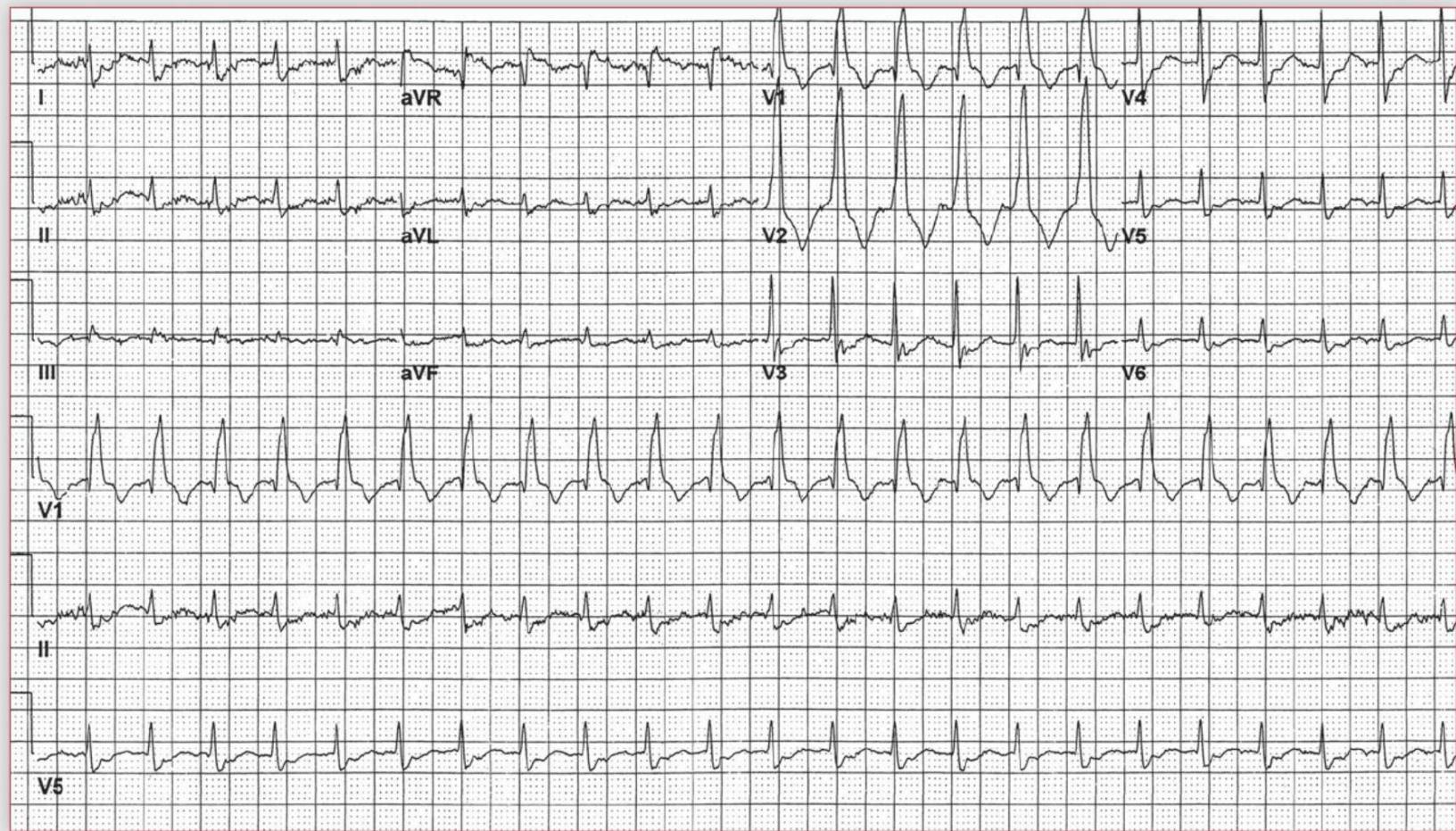
but has not yet received a diagnosis. She brings a copy of her ECG to the visit (ECG 40A). Two additional ECGs are obtained while she is tachycardic (ECGs 40B and 40C).

ECG 40B



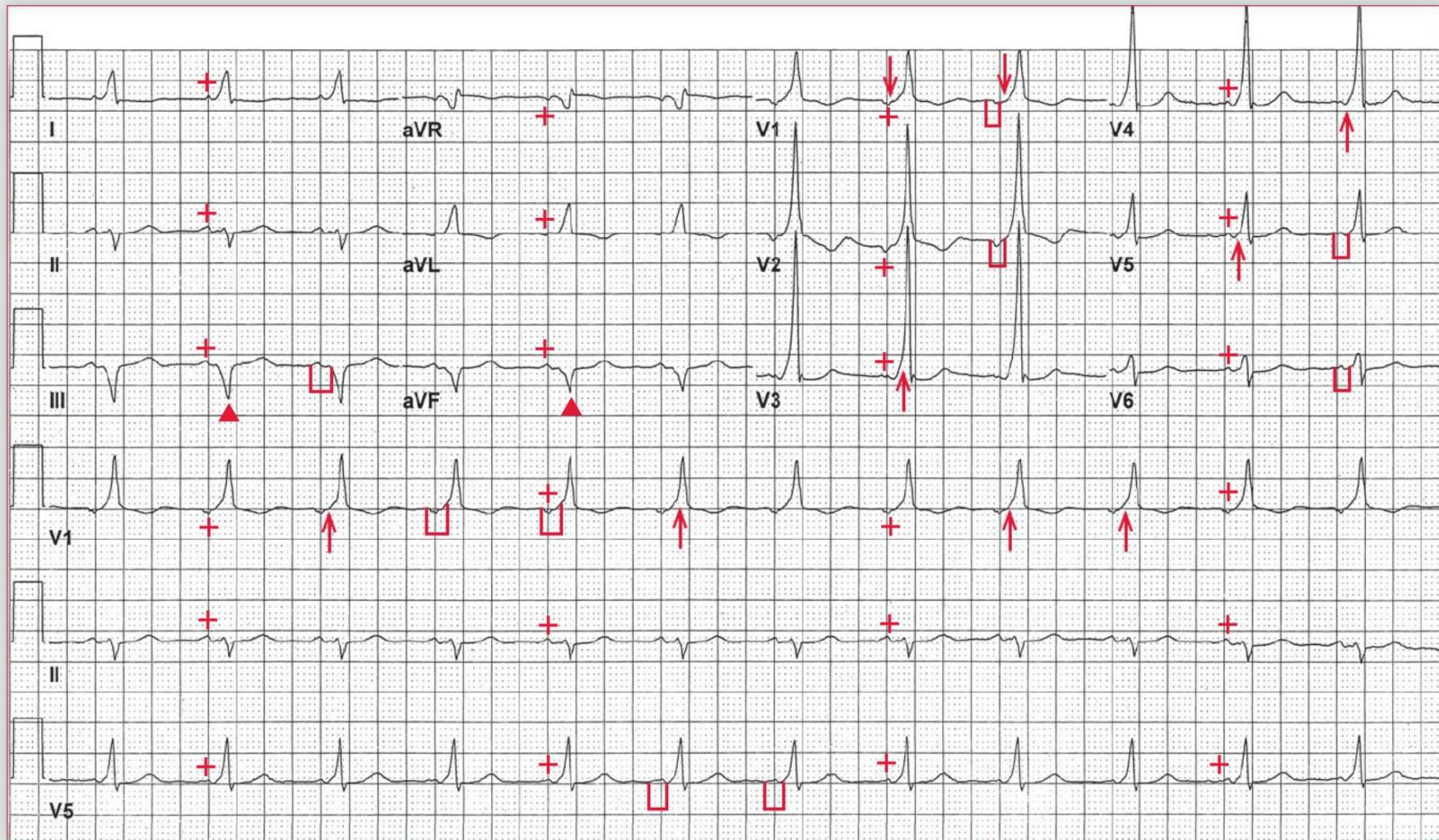
Core Case 40

ECG 40C



What does ECG 40A show?

What do ECGs 40B and 40C show?

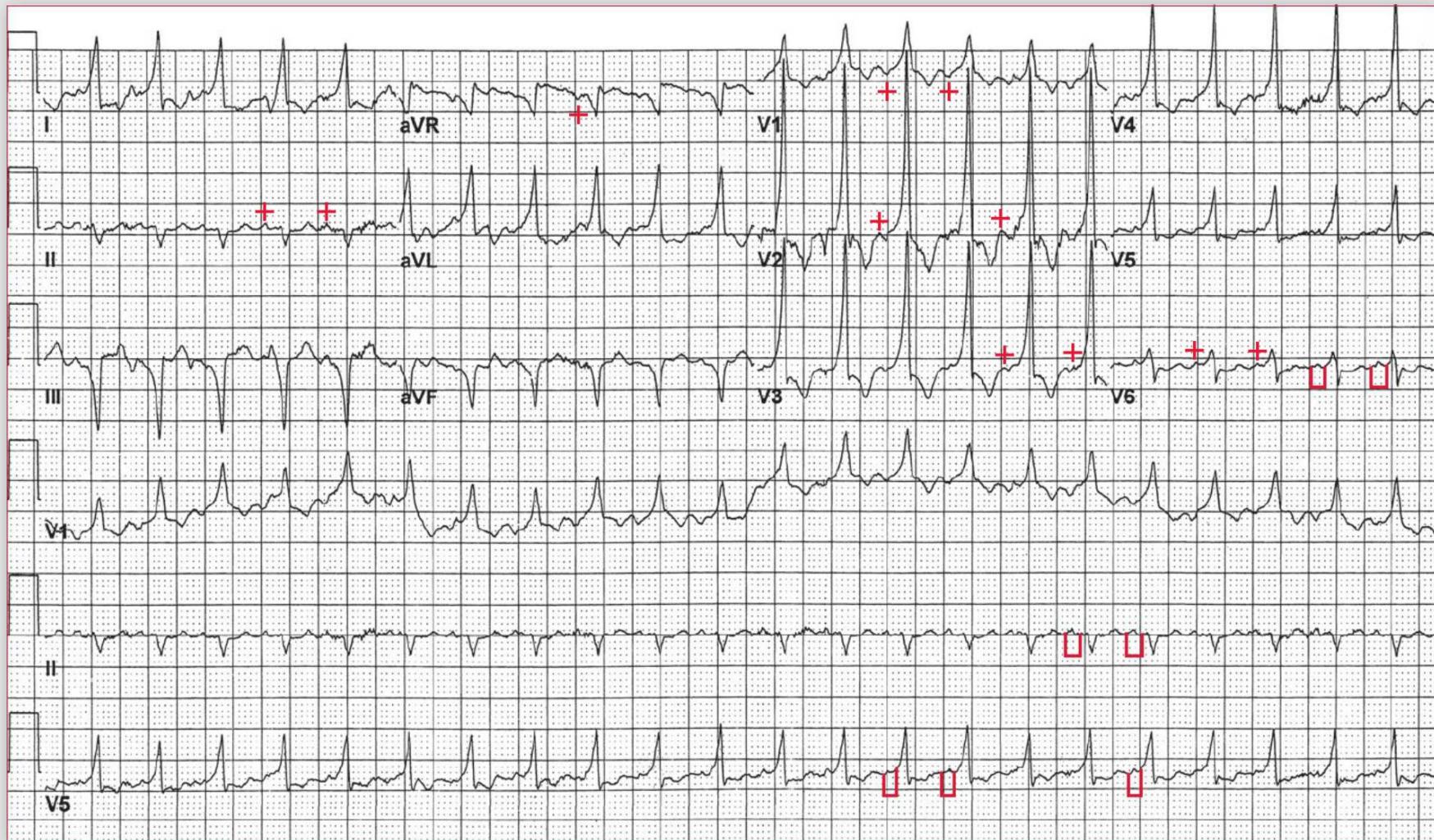


ECG 40A Analysis: Normal sinus rhythm, Wolff-Parkinson-White (WPW) pattern

In ECG 40A the rhythm is regular at a rate of 74 bpm. There is a P wave (+) before each QRS complex with a constant but short PR interval (0.12 sec) (□). The QRS complex duration is prolonged (0.14 sec) with a prominent slurred upstroke (↑) accounting for the wide QRS complex. This is a delta wave and, along with the short PR interval, is diagnostic for Wolff-Parkinson-White (WPW) pattern. Noted also is a positive delta wave in lead V1 (↓), which indicates that the accessory pathway is in the left ventricle with forces directed anteriorly toward lead V1 (WPW type A or anterior), and Q waves in leads III and aVF (▲), consistent with a pseudo inferior wall myocardial

infarction pattern. It is termed a pseudo infarction pattern because in WPW and direct myocardial activation infarction and other ventricular abnormalities cannot be diagnosed reliably. Hence there is a left-sided posteroseptal bypass pathway. There also is positive concordance across the precordium (tall R waves in leads V1-V6), which is seen whenever there is direct myocardial activation (*ie*, WPW, ventricular QRS complex, or paced complex). The QT/QTc intervals are prolonged (450/500 msec) but are normal when corrected for the prolonged QRS complex duration (390/430 msec).

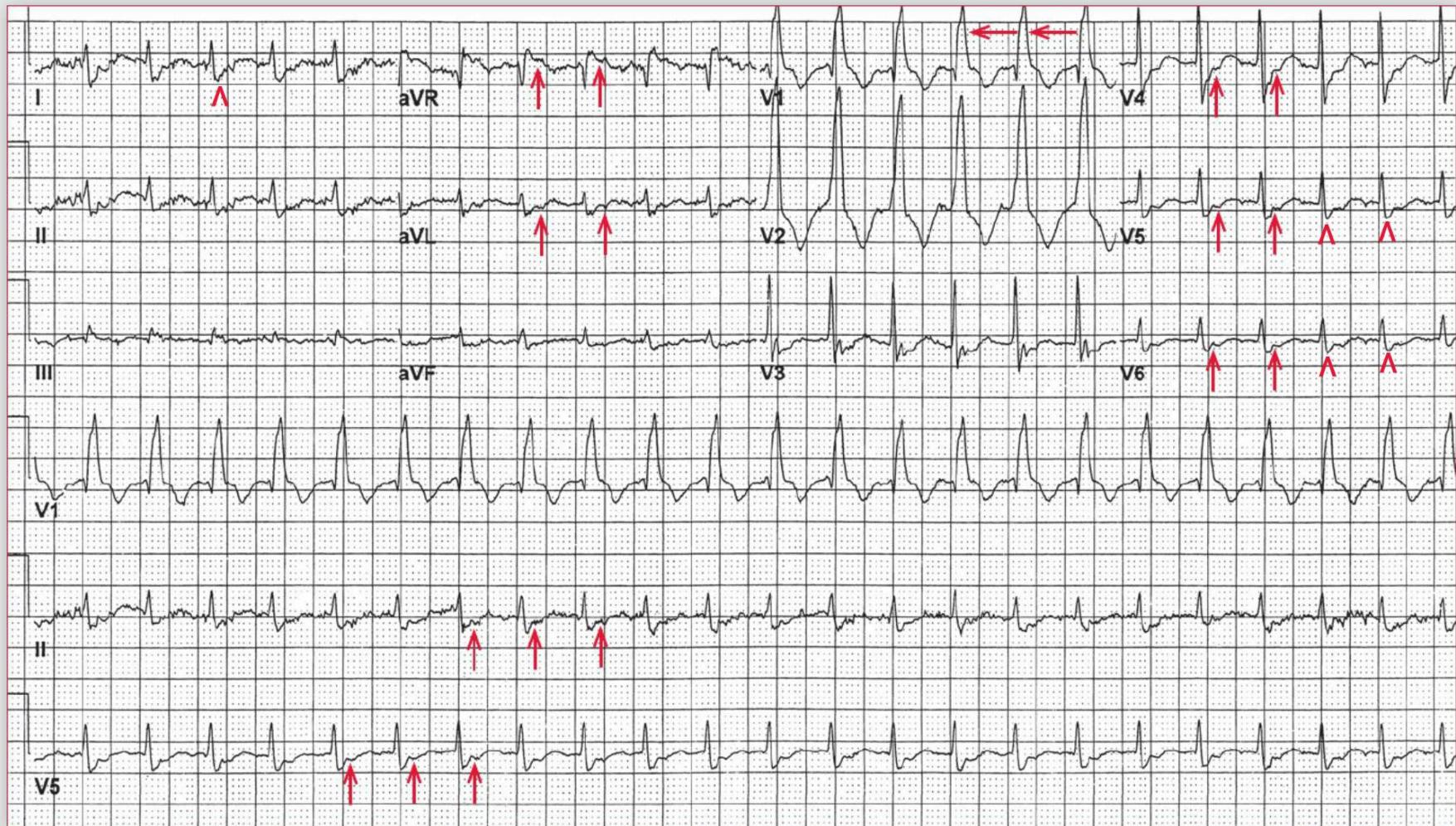
continues



ECG 40B Analysis: Sinus tachycardia, WPW pattern

ECG 40B shows a regular rhythm at a rate of 140 bpm. The QRS complex duration and morphology are identical to those seen in ECG 40A (*ie*, WPW pattern is present). The QT/QTc intervals are normal (280/430 msec and 240/370 msec when corrected for the prolonged QRS complex duration). P waves (+) can be seen in front of each QRS complex, primarily in leads V1, V3, V6, and aVR. The P wave is positive in leads II and V5-V6; therefore, the rhythm is sinus tachycardia. The PR interval is constant (0.10 sec) (□). Hence this is sinus tachycardia with WPW pattern.

continues



ECG 40C Analysis: Orthodromic AV reentrant tachycardia, rate-related
(or preexisting) right bundle branch block aberrancy

ECG 40C shows a regular rhythm at a rate of 146 bpm. In contrast to ECG 40B, there are no P waves before any QRS complex and no obvious P waves seen after the QRS complexes, although there is regular notching of the initial part of the ST segment (\uparrow), best seen in leads II, aVR, aVL, and V5-V6. As the ST segment should be smooth, notches suggest superimposed P waves. Thus, these are probable retrograde P waves and hence this is short RP tachycardia. Since the baseline ECG shows a pattern of WPW, this rhythm is AV reentrant tachycardia (AVRT).

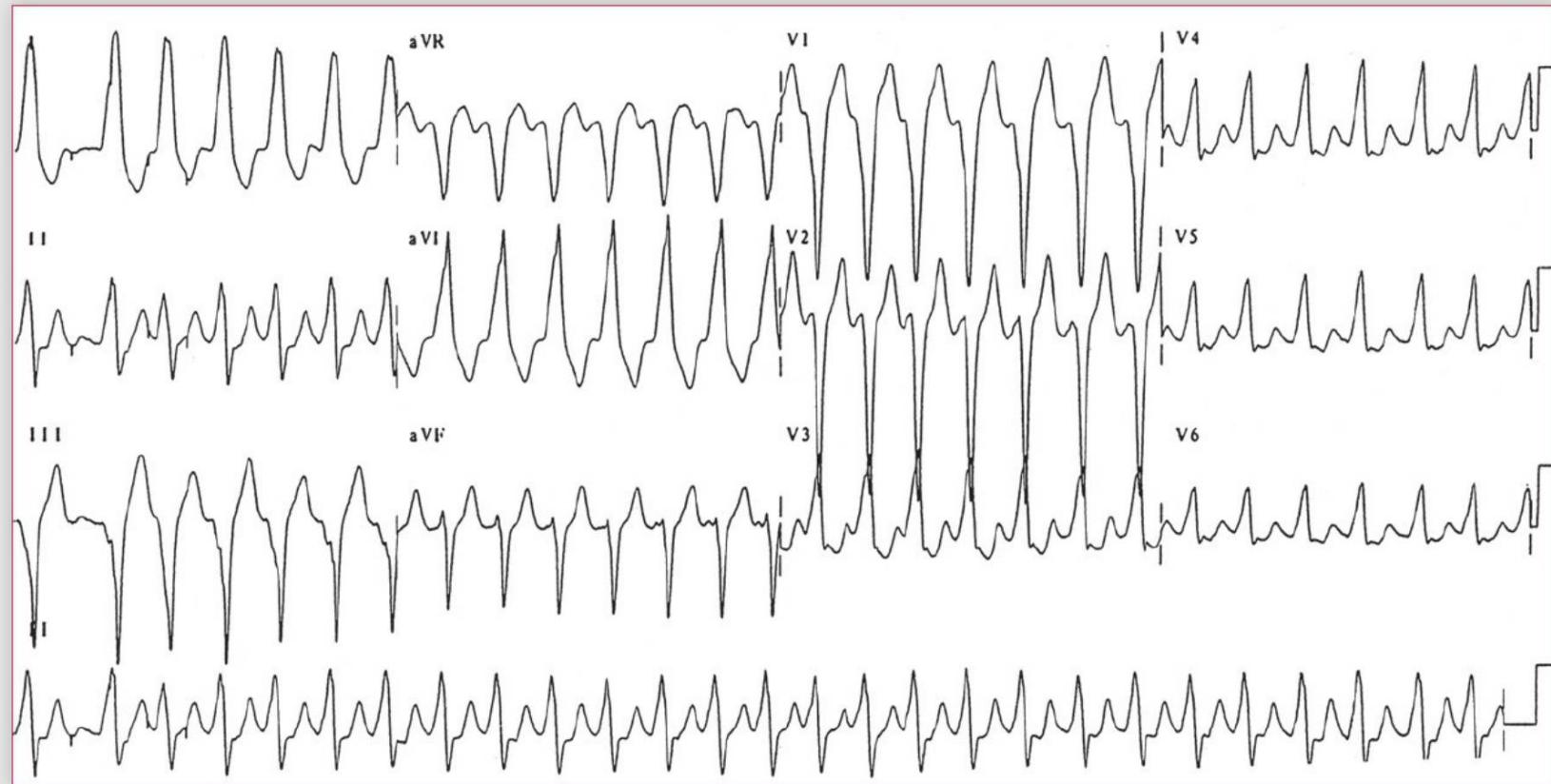
Of note, the QRS complex duration is prolonged (0.12 sec). The QT/QTc intervals are prolonged (320/500 msec) but are normal when corrected for the prolonged QRS complex duration (280/440 msec). There is an RSR' complex morphology in lead V1 (\leftarrow) and a broad S wave in leads I and V4-V6 (\wedge), a pattern of typical right bundle branch block (RBBB). Although this is a wide complex AVRT, the QRS complex has a typical RBBB morphology and the QRS complex morphology is different than the preexcited QRS complex morphology seen during normal sinus rhythm (ECG 40A). Although this is a wide complex

AVRT, it is not antidromic. In antidromic AVRT, antegrade activation of the ventricles is via the accessory pathway and retrograde activation of the atria is via the normal His-Purkinje system and AV node. The QRS complex of the tachycardia is preexcited and hence wide and it is identical in morphology to that of the preexcited complex during sinus rhythm, although it is often wider (as it is maximally preexcited). Hence this rhythm is orthodromic AVRT with a rate-related (or possibly preexistent underlying) RBBB aberrancy. With orthodromic AVRT the QRS complex is narrow and normal as activation of the ventricle is via the AV node–His-Purkinje system, while the retrograde limb is the accessory pathway resulting in negative atrial activation or a retrograde P wave. In this case the initial depolarization is indeed normal, while the terminal portion of the QRS complex is wide, a result of the RBBB aberration. As the AV node is part of the circuit for this arrhythmia, any agent that alters AV nodal conduction will be useful for terminating this arrhythmia (*ie*, adenosine, β -blocker, calcium-channel blocker, or digoxin). A vagal maneuver, such as carotid sinus pressure or Valsalva, may also be effective. ■

Core Case 41

You are called to assess a patient in the emergency department who has a wide complex tachycardia (ECG 41A). The patient reports palpitations but is hemodynamically stable. The patient's baseline ECG (41B) is obtained after termination of the tachycardia.

ECG 41A

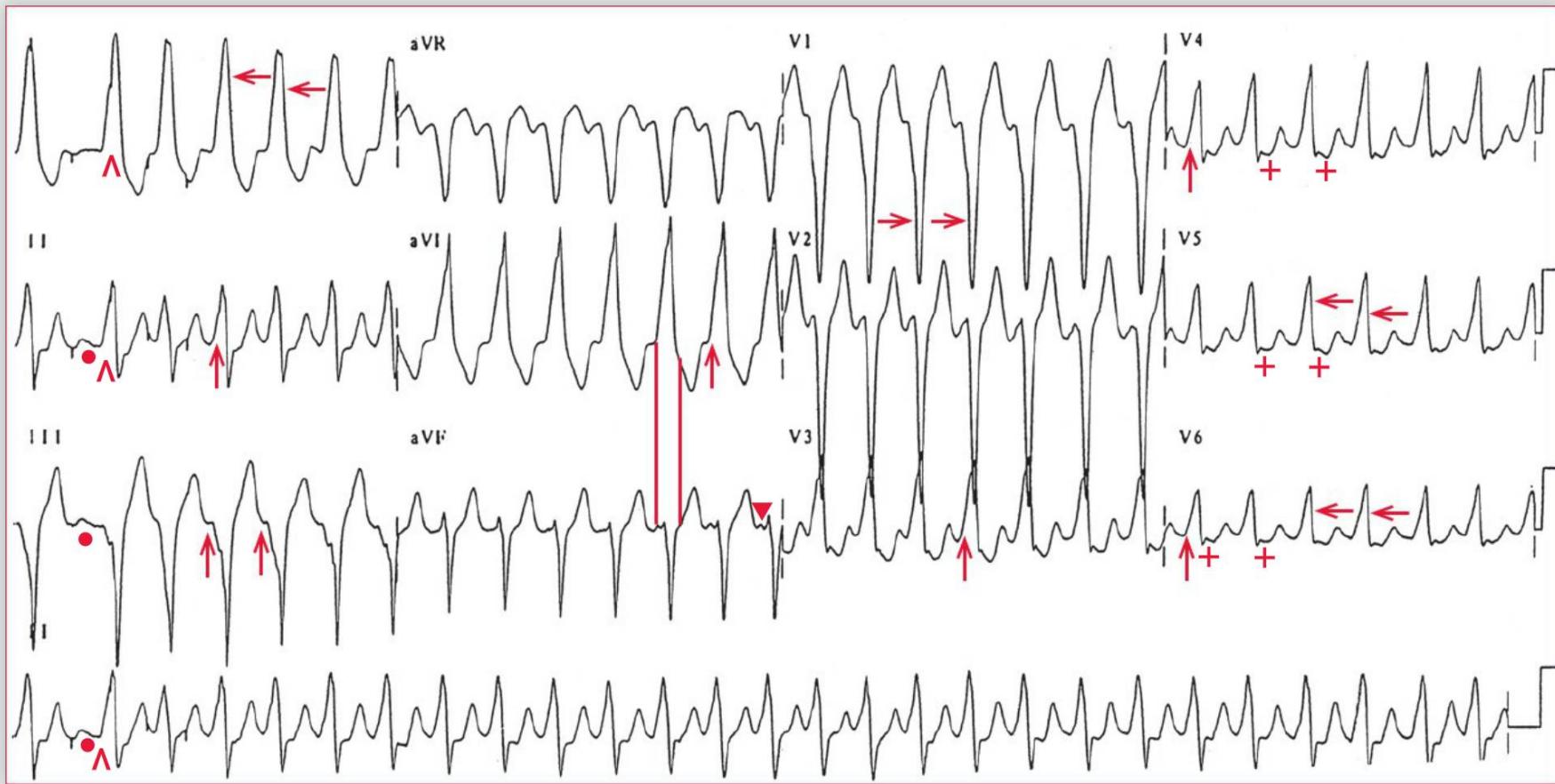


What is the diagnosis?

What acute therapy would be appropriate?

ECG 41B





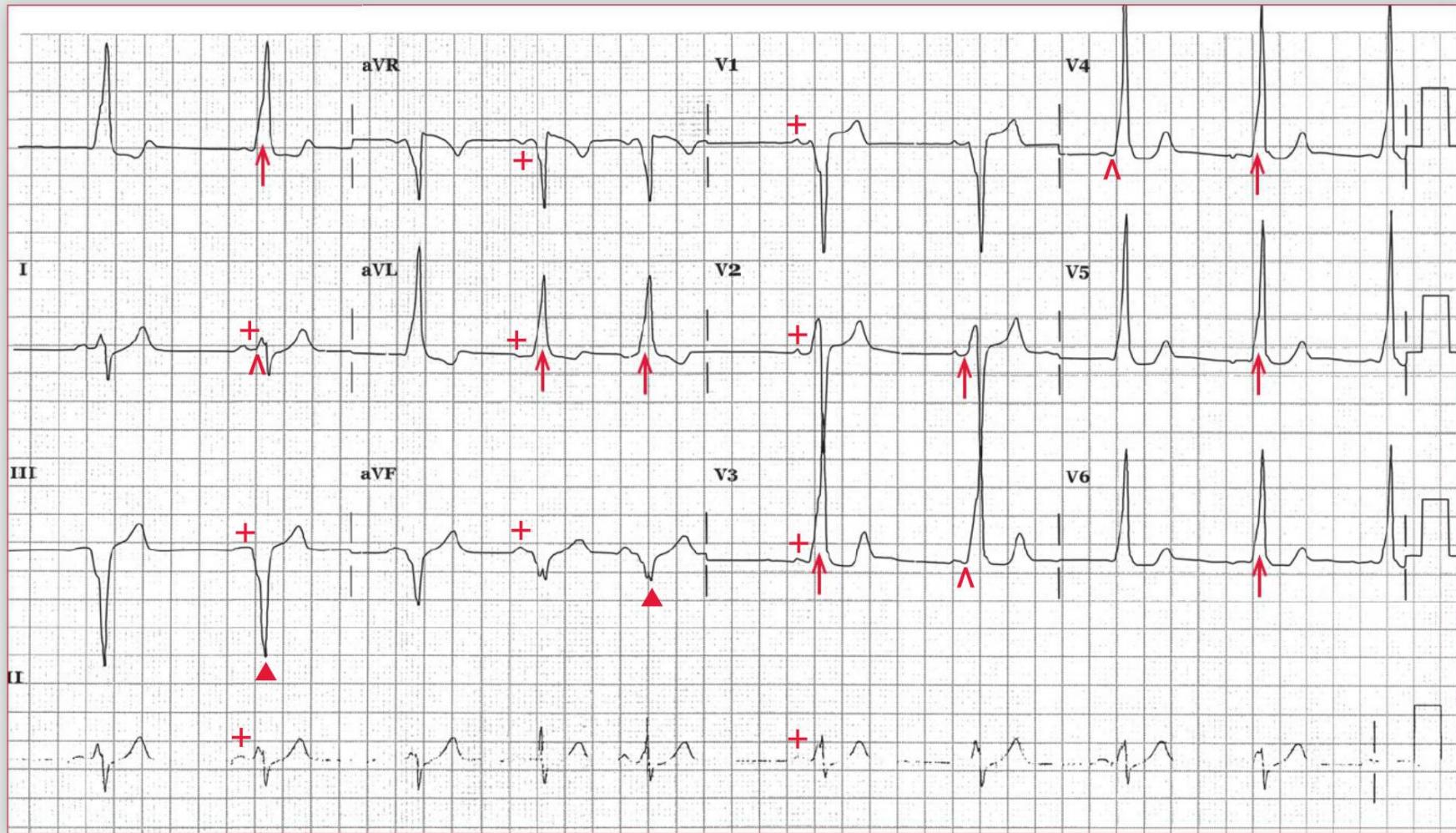
ECG 41A Analysis: Antidromic AV reentrant tachycardia

ECG 41A shows a regular rhythm at a rate of 160 bpm. No P waves are seen before any QRS complex, and there are no obvious P waves after the QRS complexes. It should be noted that there are small waveforms before the QRS complexes in lead aVF (▼); however, when comparing the QRS width in this lead with the QRS width in other leads, (eg, lead aVL [||]), it can be seen that these waveforms are part of the QRS complex. There are small notches after the QRS complex in leads V4-V6 (+); these have a fixed relationship to the QRS complexes and are possibly P waves. If they are P waves, this would be short RP tachycardia. The QRS complex duration is prolonged (0.16 sec), and there is a left bundle branch block morphology (tall R wave in leads I and V5-V6 [←] and a QS complex in lead V1 [→]). The QT/QTc intervals are prolonged (320/520 msec) but are normal when corrected

for the prolonged QRS complex duration (240/390 msec). The etiology of the wide complex tachycardia is not certain; it may be ventricular tachycardia or supraventricular tachycardia with aberration.

Of note is the second QRS complex (Λ), which is preceded by a P wave (●). The PR interval is 0.20 second, although the PR segment is short and there is a broad P wave accounting for the normal PR interval. The morphology of the QRS complex after the P wave is identical to the QRS complex morphology during the tachycardia. This indicates that the wide complex tachycardia is likely supraventricular in origin. It appears to have a prolonged upstroke (↑), suggesting (but not proving) a delta wave and hence Wolff-Parkinson-White (WPW) pattern.

continues



ECG 41B Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern

In ECG 41B the rhythm is regular at a rate of 86 bpm. There is a P wave (+) before each QRS complex with a constant PR interval (0.16 sec), although the PR segment is short (Δ) and there is a broad P wave accounting for the normal PR interval. The PR interval is identical to that of the second QRS complex in ECG 41A. The QRS complex has a slurred upstroke (↑) that is a delta wave and hence this is WPW pattern (short RP segment and wide QRS complex with a delta wave). Since the QRS morphology of the tachycardia in each lead of ECG 41A is identical to that during sinus rhythm (wide and preexcited) and WPW pattern is present, the arrhythmia is antidromic AV reentrant tachycardia (AVRT). The presence of Q waves in leads III and aVF (▲) localizes the accessory pathway to the posteroseptal area; the negative delta wave in lead V1 localizes it to the right ventricle as the initial impulse is directed away from lead V1 or posteriorly (termed WPW type B or back). The QT/QTc intervals are prolonged (460/550 msec) but are normal when corrected for the prolonged QRS complex duration (400/450 msec).

A wide complex tachycardia may be either ventricular tachycardia or supraventricular tachycardia with aberration. The aberration may be due to a rate-related or functional bundle branch block, a preexisting aberrated complex at baseline (right or left bundle branch block pattern), or preexcitation. As one etiology may be ventricular tachycardia, AV nodal blocking agents should not be given. They are not effective for terminating a ventricular tachycardia and may indeed be harmful, as transient hypotension may provoke ventricular fibrillation. However, if

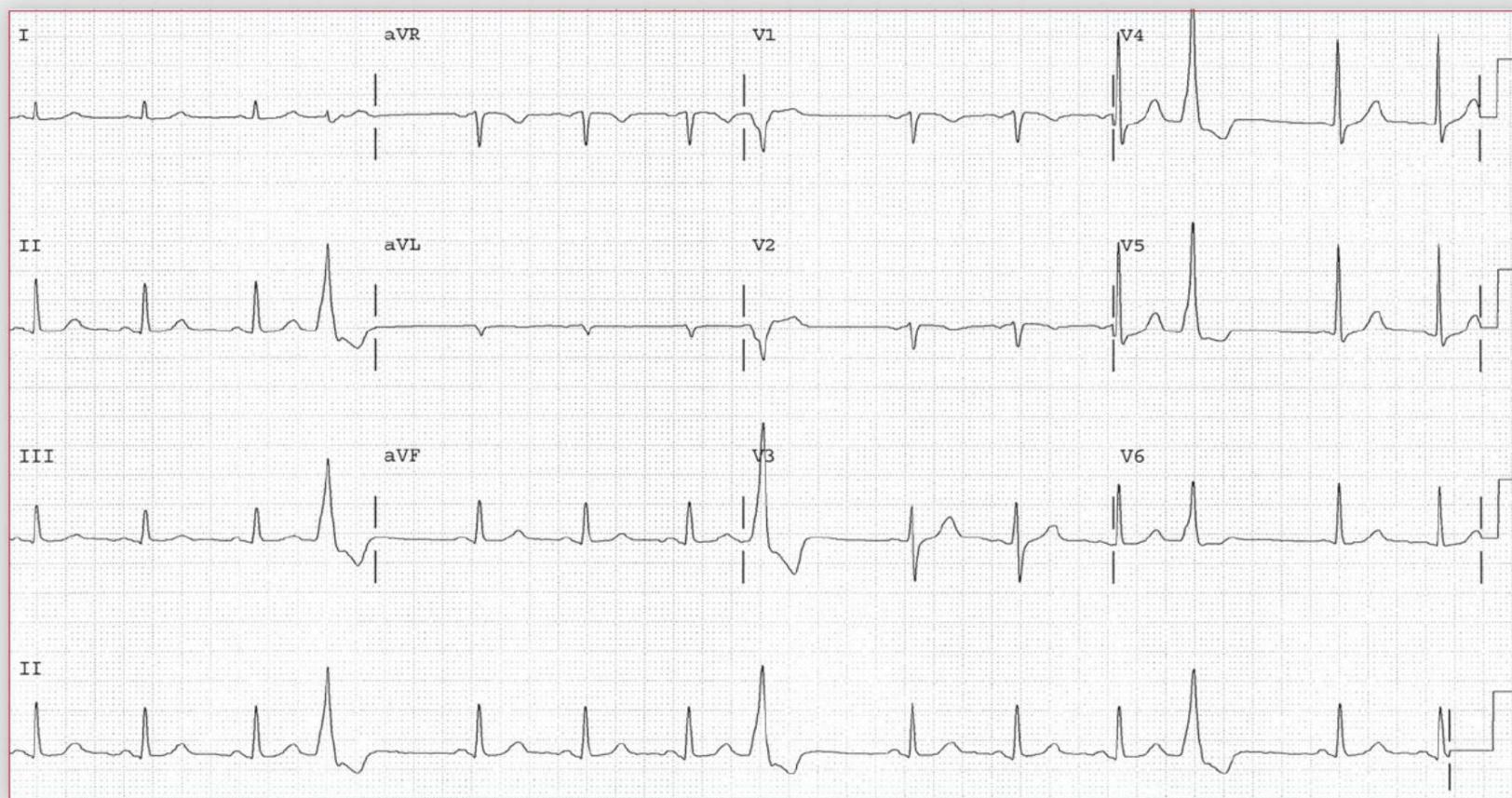
the etiology is supraventricular or junctional tachycardia, an AV nodal blocking agent will be effective as the AV node is part of the reentrant circuit. In AV nodal reentrant tachycardia (AVNRT), the reentrant circuit is entirely within the AV node (*ie*, due to dual AV nodal pathways). In AVRT the AV node is part of a much larger circuit involving the accessory pathway, atrial and ventricular myocardium, and AV node-His-Purkinje system. Altering the electrophysiologic properties of any part of this circuit will likely terminate an AVRT. As the AV node is the site of slowest conduction, producing changes in AV nodal properties or AV blockade is most likely to terminate the arrhythmia.

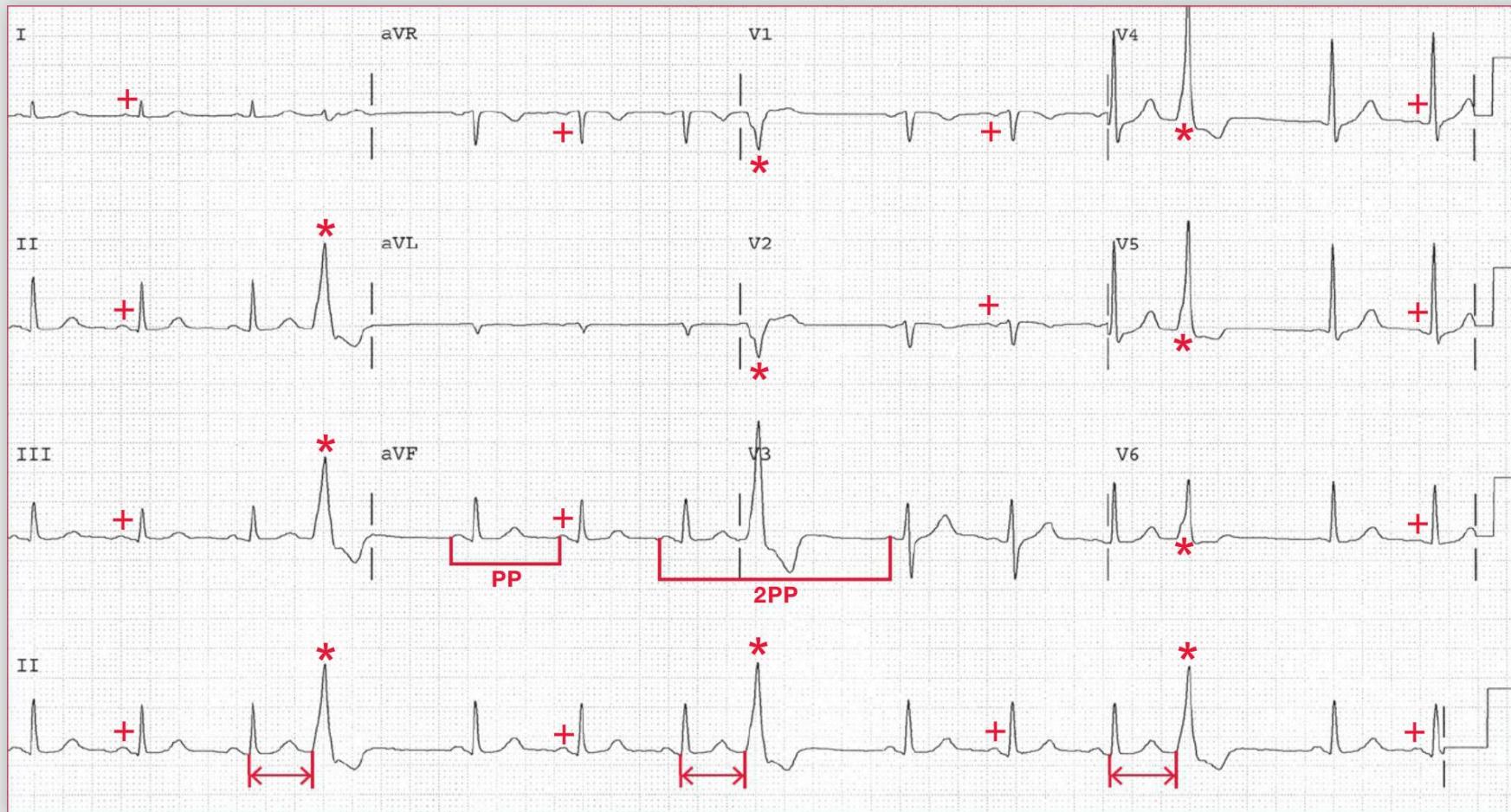
With an antidromic AVRT, the antegrade activation of the ventricular myocardium is via the accessory pathway while retrograde activation of the atria is via the normal His-Purkinje system and AV node. Hence the QRS complex will be wide and preexcited. The QRS complex of the tachycardia will have the same morphology (and delta wave direction) as the preexcited complex during sinus rhythm, although it may be wider as it is maximally preexcited (*ie*, all ventricular activation is via the accessory pathway and there is no fusion as may be seen with a sinus complex). This is the most important way to establish antidromic AVRT. Therefore, the treatment of antidromic AVRT is similar to that for orthodromic AVRT (*ie*, vagal enhancement or AV nodal blocking agents). The important issue is establishing that the etiology of the wide complex tachycardia is supraventricular arrhythmia or antidromic AVRT and not ventricular tachycardia. ■

Notes

A 55-year-old man is seen in the emergency department for epistaxis. On physical exam, he is incidentally noted to have an irregular pulse, which occasionally appears to "drop or skip a beat."

What is causing these dropped or skipped beats?
Is any therapy warranted?





ECG 42 Analysis: Sinus rhythm, unifocal premature ventricular complexes

The rhythm is regularly irregular as a result of three premature and wide QRS complexes (*) (0.18 sec). The morphology of these wide complexes is unusual, and they do not have a typical right or left bundle branch block. They are not preceded by a P wave and are followed by a pause. The coupling interval of these three early QRS complexes (the interval between the premature complex and the preceding QRS complex) is the same; that is, there is a fixed coupling interval (\leftrightarrow). These are premature ventricular complexes (PVCs). They all have the same morphology and hence are unifocal.

All of the narrower QRS complexes are regular at a rate of 82 bpm. The axis is normal, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. The QT/QTc intervals are normal (380/440 msec). There is a P wave (+) preceding each QRS complex, and the PR interval is constant (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm.

The PVCs are followed by a full compensatory pause; that is, the PP interval surrounding the PVC is equal to two sinus PP intervals (⊍).

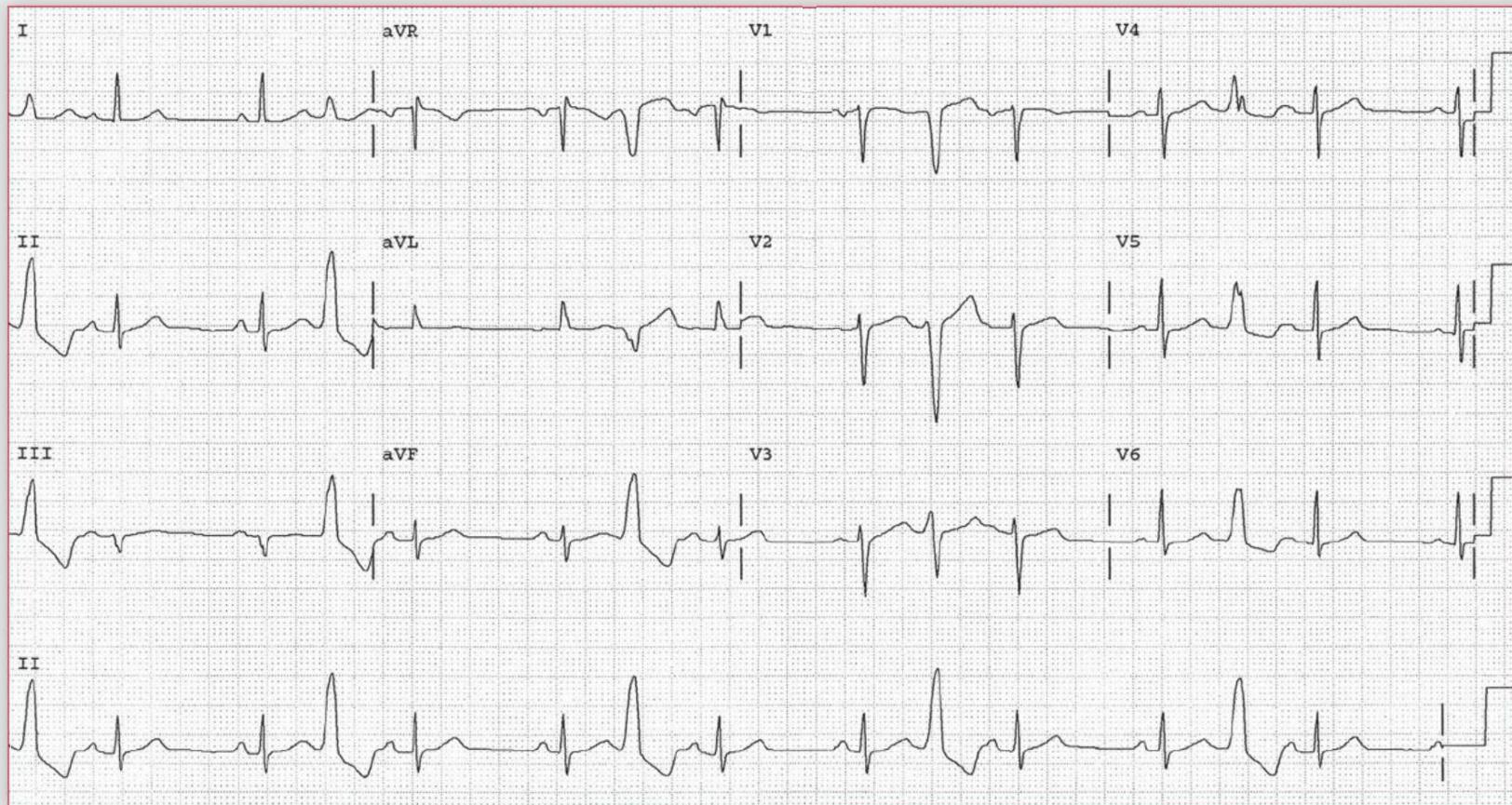
This is due to the fact that the PVC retrogradely penetrates and completely depolarizes the AV node, blocking the antegrade conduction of the subsequent sinus beat. However, the next sinus beat can be conducted through the AV node on time. Thus one sinus beat is blocked and missing, accounting for two PP intervals around the PVC.

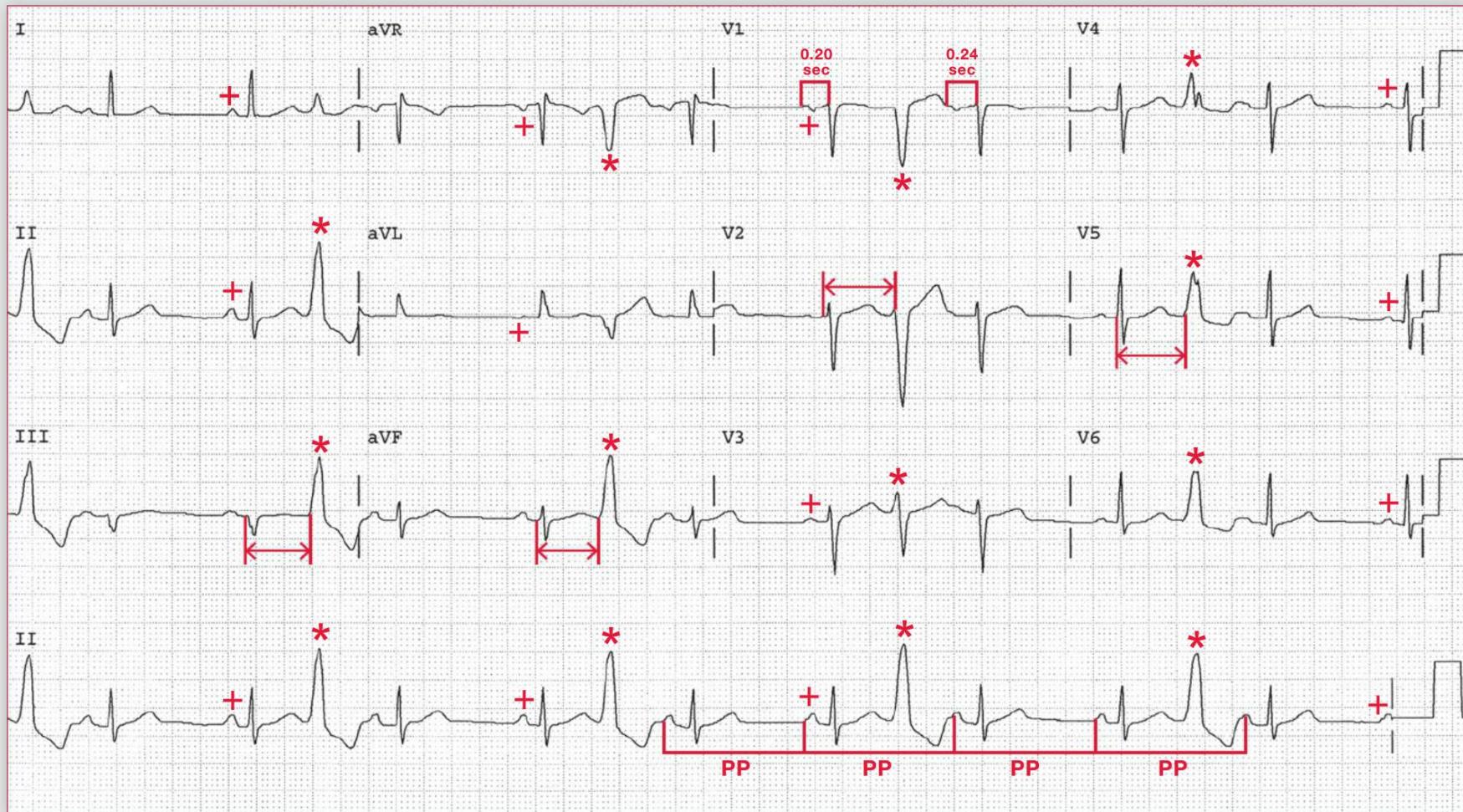
PVCs are common and may occur in up to 90% of the population, although they are generally infrequent in most people. They are usually benign and do not require therapy. However, they may cause symptoms, particularly palpitations, which may be debilitating. The palpitations are the result of the pause and post-extrasystolic potentiation due to the increased inotropy that occurs when there is an increase in systolic volume (as occurs with the pause). This is the result of the Frank-Starling effect. If they do cause symptoms, therapy may be necessary for symptom relief. Often a β -blocker is effective for symptom relief by reducing post-extrasystolic inotropy, but it does not generally reduce the frequency of the premature complexes. A mild tranquilizer may be useful for allaying the anxiety associated with this rhythm disturbance. Infrequently, an antiarrhythmic drug will be needed for arrhythmia suppression. ■

Notes

A 32-year-old woman takes her pulse and notes that there is some irregularity. She feels well but is concerned that there is something wrong with her heart. She asks you to obtain an ECG.

**What does the ECG show?
Is any therapy necessary?**





ECG 43 Analysis: Normal sinus rhythm, interpolated unifocal premature complexes, ventricular trigeminy, retrograde concealed conduction

There is a regularly irregular rhythm as a result of multiple premature complexes (*) in a repeating pattern (three QRS complexes and a pause). These premature complexes are wide (0.18 sec) and abnormal in morphology (*ie*, they do not have a typical right or left bundle branch block morphology) and are not preceded by a P wave. These are, therefore, premature ventricular complexes (PVCs) and they all have the same morphology (*ie*, they are unifocal). As there is a repeating pattern of every third complex being a PVC, this is termed ventricular trigeminy. There is a fixed coupling interval (↔) between the PVC and the complex before it. This is the result of a reentrant mechanism within the ventricular myocardium and Purkinje system that is responsible for the premature beat, with the impulse circulating around the circuit once. As the conduction velocity around the circuit is stable, there is a fixed coupling interval.

The two narrow QRS complexes that surround the PVC have a normal duration (0.08 sec) and morphology with a physiologic left axis, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (400/400 msec). There is a P wave (+) before each of these narrow complexes. The P waves are positive in leads I, II, aVF, and V4-V6; hence this is a normal sinus rhythm. The PP interval is stable (□), and the

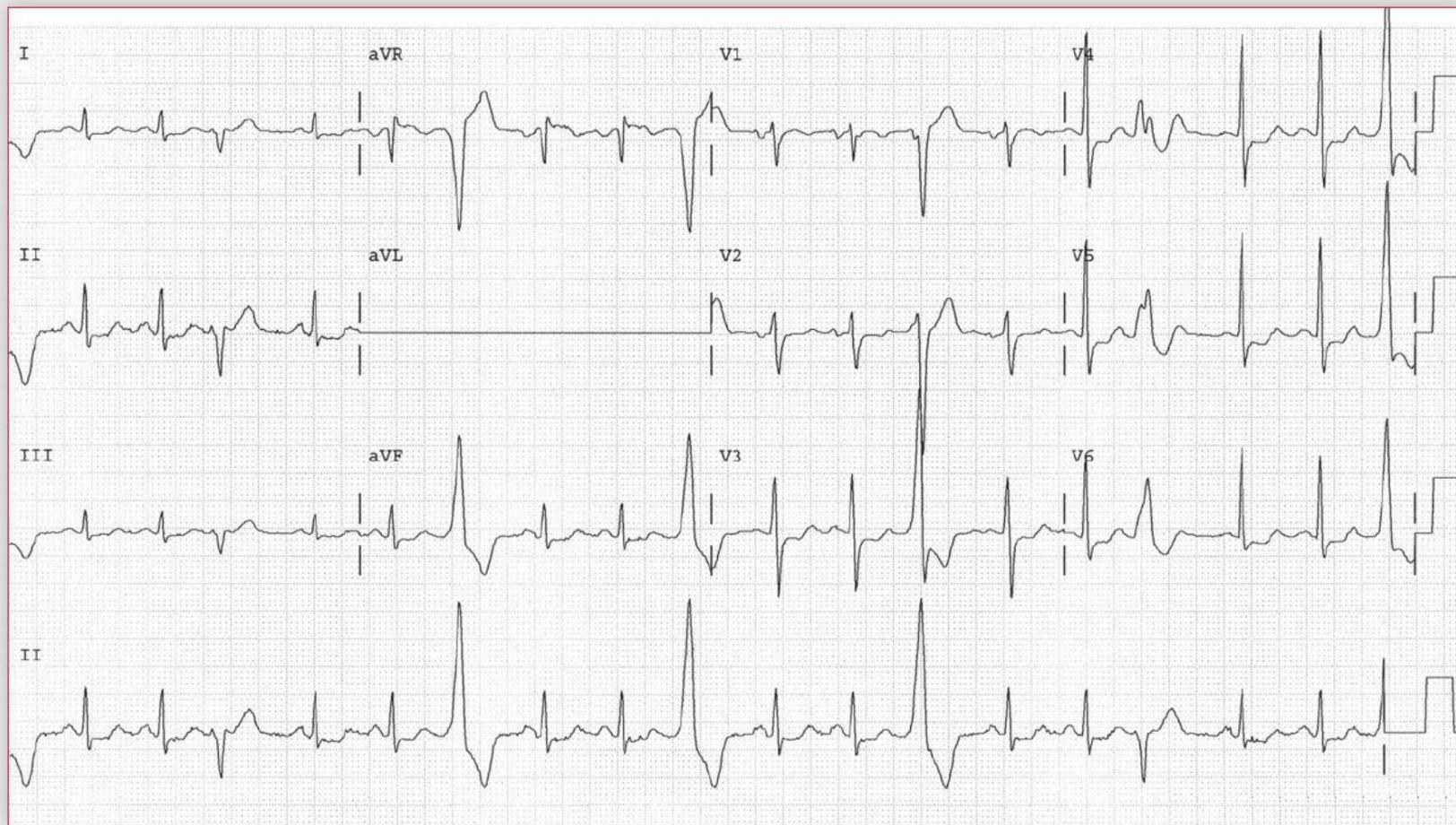
sinus rate is 60 bpm. As the PVC does not alter the underlying sinus rate (PP interval), the PVCs are termed interpolated PVCs. Since every third QRS complex is a PVC, this is ventricular trigeminy. Although the two narrow QRS complexes are preceded by a P wave, the PR intervals are not the same. The PR interval of the sinus beat following the PVC is slightly longer (0.24 sec) (□) than the PR interval (0.20 sec) of the sinus beat before the PVC. This is a result of retrograde concealed conduction, which is frequently seen with interpolated PVCs. The PVC results in retrograde conduction through the AV node. However, an appropriately timed premature complex may not completely penetrate the AV node but rather is extinguished within the node, partially depolarizing it (*ie*, it is concealed within the AV node). Hence the AV node is partially refractory. In this situation the on-time sinus beat may be conducted through the AV node in an antegrade direction but at a slower conduction velocity as the AV node is partially refractory due to the incomplete retrograde conduction from the premature complex.

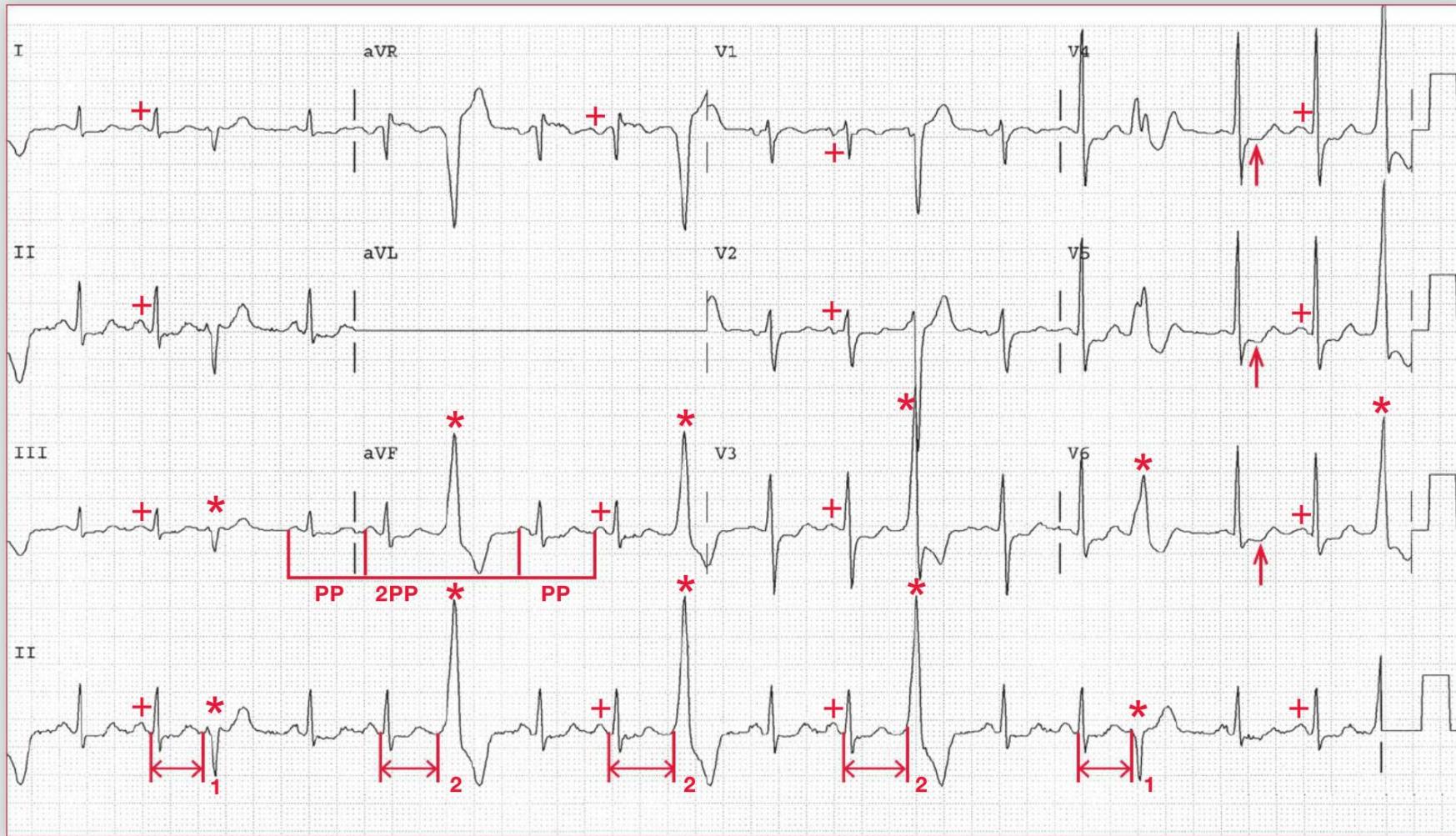
The presence of ventricular trigeminy indicates that there is a repeating pattern to the arrhythmia. It does not have any additional importance aside from the fact that there are frequent PVCs. Generally no therapy is needed unless there are symptoms that are disturbing. ■

Notes

A 71-year-old man is admitted for a non-ST-segment elevation myocardial infarction. He is noted to have an irregular heart rhythm.

What is the etiology of his irregular heartbeat?





ECG 44 Analysis: Sinus tachycardia, multifocal premature ventricular complexes in a trigeminal pattern, ST-segment depression

There is a regularly irregular rhythm, a result of several premature QRS complexes (third, sixth, ninth, 12th, 15th, and 18th) (*). These premature complexes are wide (0.18 sec), have an abnormal morphology that does not resemble either a typical right or left bundle branch block, and are not preceded by a P wave. These are premature ventricular complexes (PVCs). The coupling interval (↔) between the premature complex and the preceding QRS complex is fixed, and there is a pause after each premature complex. The PP interval surrounding the premature complex is twice the underlying PP interval (□); hence these are full compensatory pauses. It is noted that the premature complexes have two different morphologies (1, 2). Therefore, these are multifocal PVCs. Since every third QRS complex is a premature complex, this is termed ventricular trigeminy.

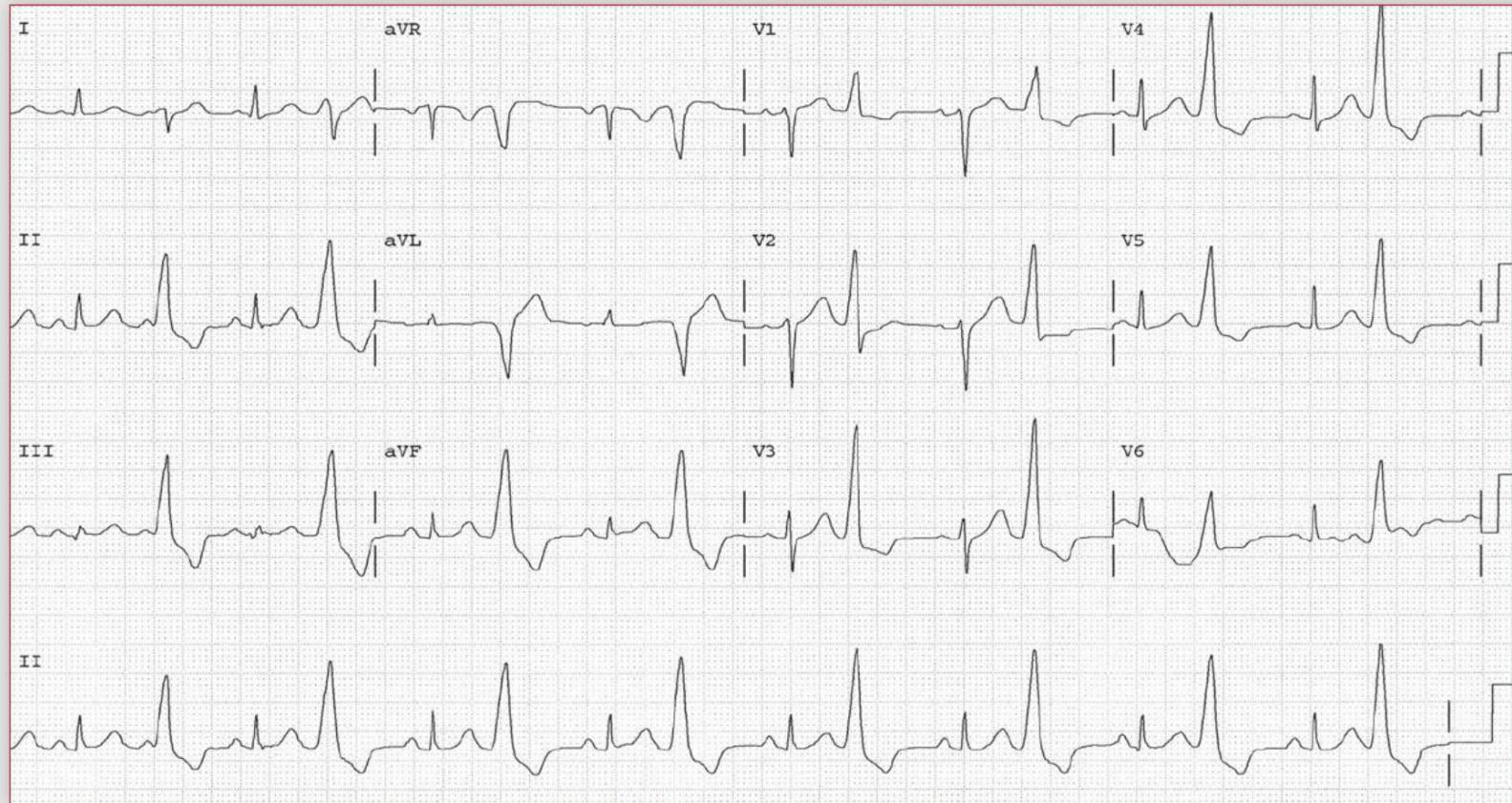
The duration of the narrow QRS complexes is 0.08 second, and they have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The rate is 104 bpm. The QT/QTc intervals are normal (340/450 msec). There is a P wave (+) before each of these QRS complexes with a stable PR interval (0.14 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia with multifocal PVCs in a trigeminal pattern.

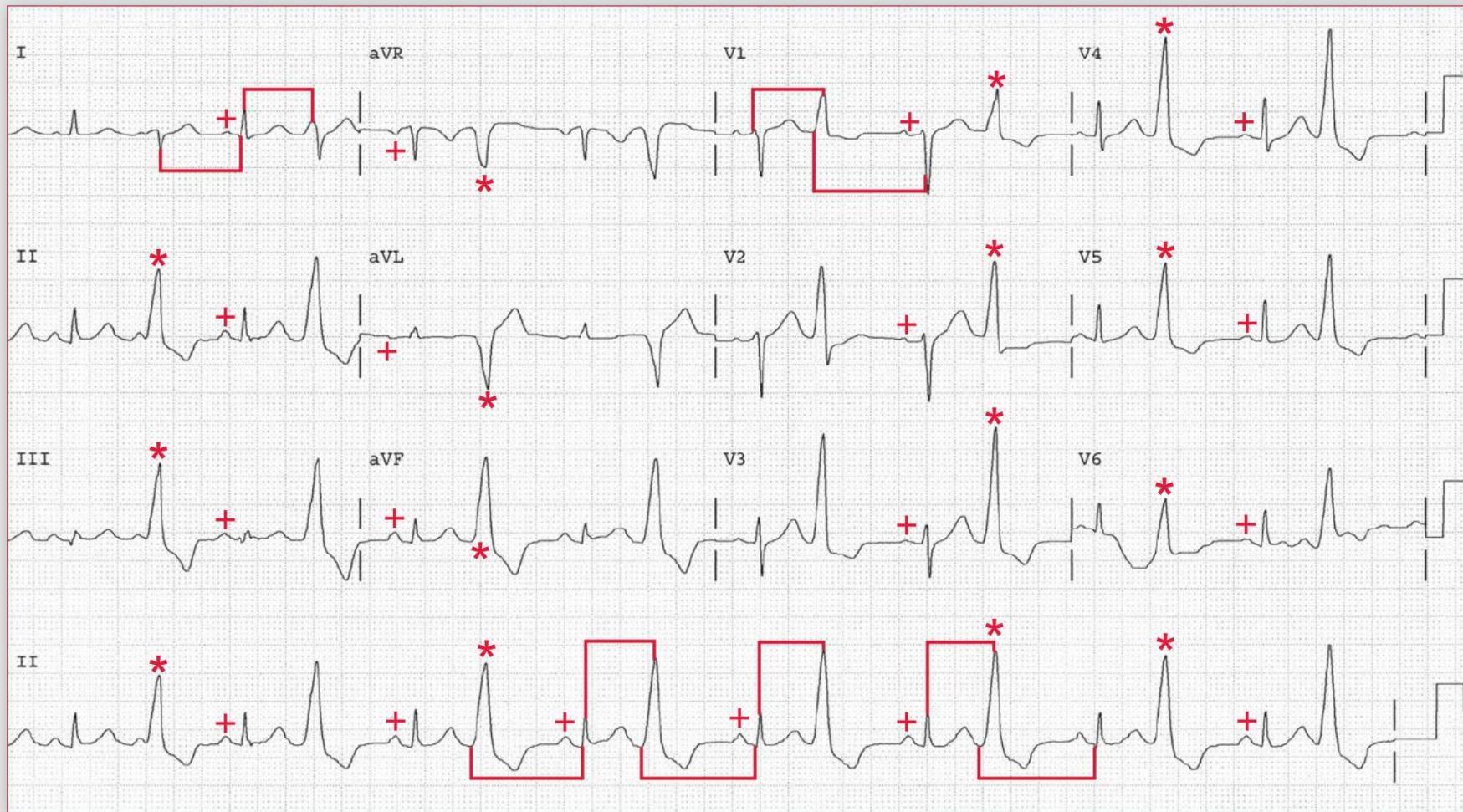
Also noted is horizontal ST-segment depression (↑), particularly in leads V3-V6. These changes are the result of subendocardial ischemia, and this is associated with the diagnosis of non-ST-segment elevation myocardial infarction. ■

Notes

A patient is noted by his nurse to have a heart rate detected by manual pulse that is about half as much as is being calculated by the telemetry monitor.

**What is occurring?
Is any therapy warranted?**





ECG 45 Analysis: Normal sinus rhythm, unifocal premature ventricular complexes in a bigeminal pattern

The rhythm is regularly irregular, and there is a repeating pattern of long and short RR intervals. All the long RR intervals (□) are the same, and all the short RR intervals (■) are the same. There are also two types of QRS complex morphologies. The narrow QRS complexes have a duration of 0.08 second and a normal morphology. There is a P wave (+) before each of these complexes. The PR interval is constant (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The PP interval is stable at a rate of 90 bpm. The QT/QTc intervals are normal (360/440 msec). After each sinus complex there is a QRS complex that is early, wide, abnormal in morphology (not resembling either a typical right or left bundle branch block), and with a duration of 0.14 second (*). There are no P waves before these wide QRS complexes. They are premature ventricular complexes (PVCs), and a pause or long RR interval follows them. Since all

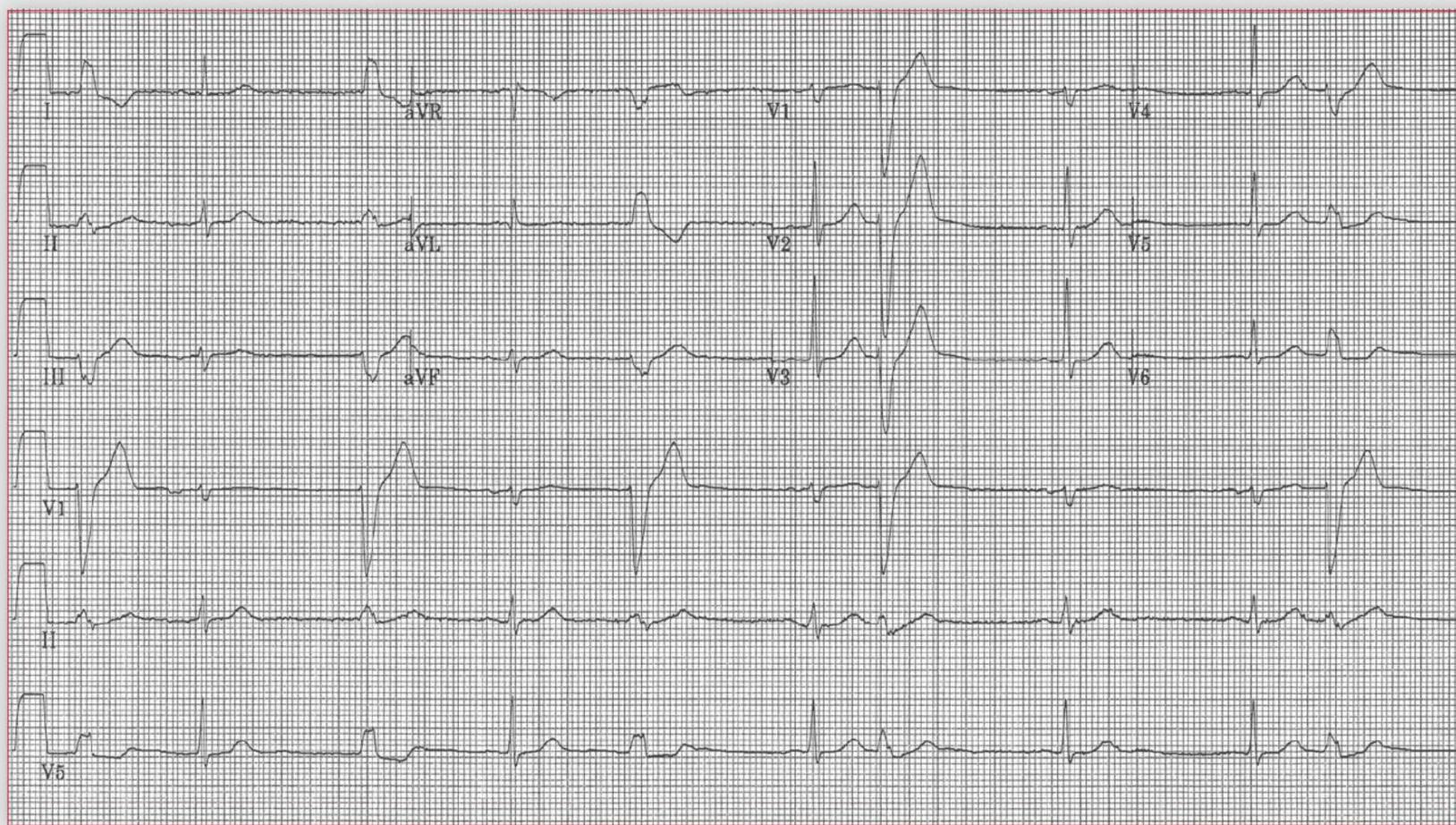
the PVCs have the same morphology, they are unifocal. There is a fixed relationship (■) between the sinus beat and the PVC (fixed coupling). As every other complex is a PVC, this is called ventricular bigeminy (*ie*, a repeating pattern with every other QRS complex being a PVC). There is no specific clinical significance associated with ventricular bigeminy; this represents only frequent premature complexes. Because the PVCs are benign, no specific therapy is needed unless there are associated symptoms. The PVC often results in a small stroke volume as a result of inadequate left ventricular diastolic filling due to the early occurrence of ventricular contraction. This may result in the absence of a palpable pulse, as occurred in this patient. Therefore, the effective pulse rate is slow and may result in symptoms related to a relative bradycardia. ■

Notes

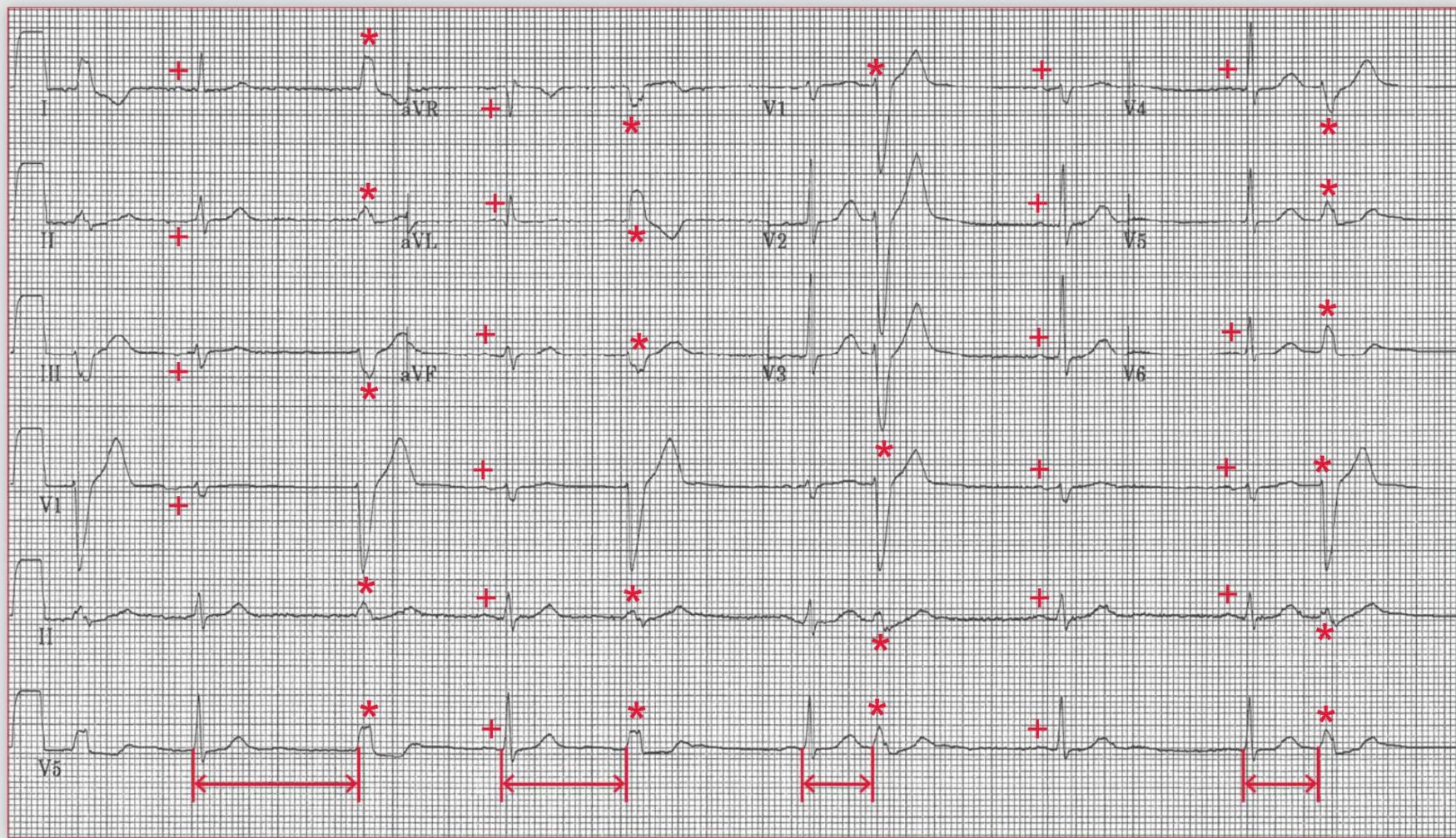
A patient is brought to the emergency department for lightheadedness, fatigue, and palpitations. She is found to have an irregularly irregular pulse. An ECG is obtained.

Based on this ECG, what is the explanation for the irregular pulse?

What more information would you need to prove your theory?



Podrid's Real-World ECGs



ECG 46 Analysis: Unifocal premature ventricular complexes, ventricular parasytole

The rhythm is irregularly irregular. The rate is approximately 60 bpm. The narrow QRS complexes (duration, 0.08 sec) are preceded by a P wave (+) that is positive in leads I, II, aVF, and V4-V6 and hence are sinus in origin. The PR interval is stable (0.18 sec). These are, therefore, sinus complexes. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec).

Following each sinus complex is a wide QRS complex (*) (duration, 0.16 sec) with an abnormal morphology that resembles neither a typical right nor left bundle branch block and that is not preceded by a P wave. There is a pause after each wide QRS complex. These are premature ventricular complexes (PVCs). Each one has the same morphology, and hence they are unifocal. However, the coupling interval (↔) between the sinus beat and the PVC is variable. Most often unifocal PVCs have a fixed relationship to the preceding sinus beat (*ie*, they have a fixed coupling interval). The presence of a fixed coupling interval indicates that the mechanism for the premature beat is reentry, which is the most common mechanism for arrhythmia. When there is a single PVC, the impulse circulates around the reentrant circuit for one cycle. Since the circuit is always the same and the conduction velocity is stable, the

coupling interval is fixed. The presence of a variable coupling interval indicates that the PVC is not the result of reentry, but is due to an ectopic focus that has its own intrinsic rate that is different than that of the sinus node. There is entrance block into this focus, so that it is not suppressed by another impulse (*ie*, sinus impulse), and exit block from this focus such that it can only be conducted into and activate the ventricular myocardium when the myocardium is not refractory (as a result of a previous sinus impulse). As a result the PVC resulting from the ectopic focus seems to occur in random fashion and hence there is a variable coupling interval between the sinus beat and the premature beat. This is termed ventricular parasystole.

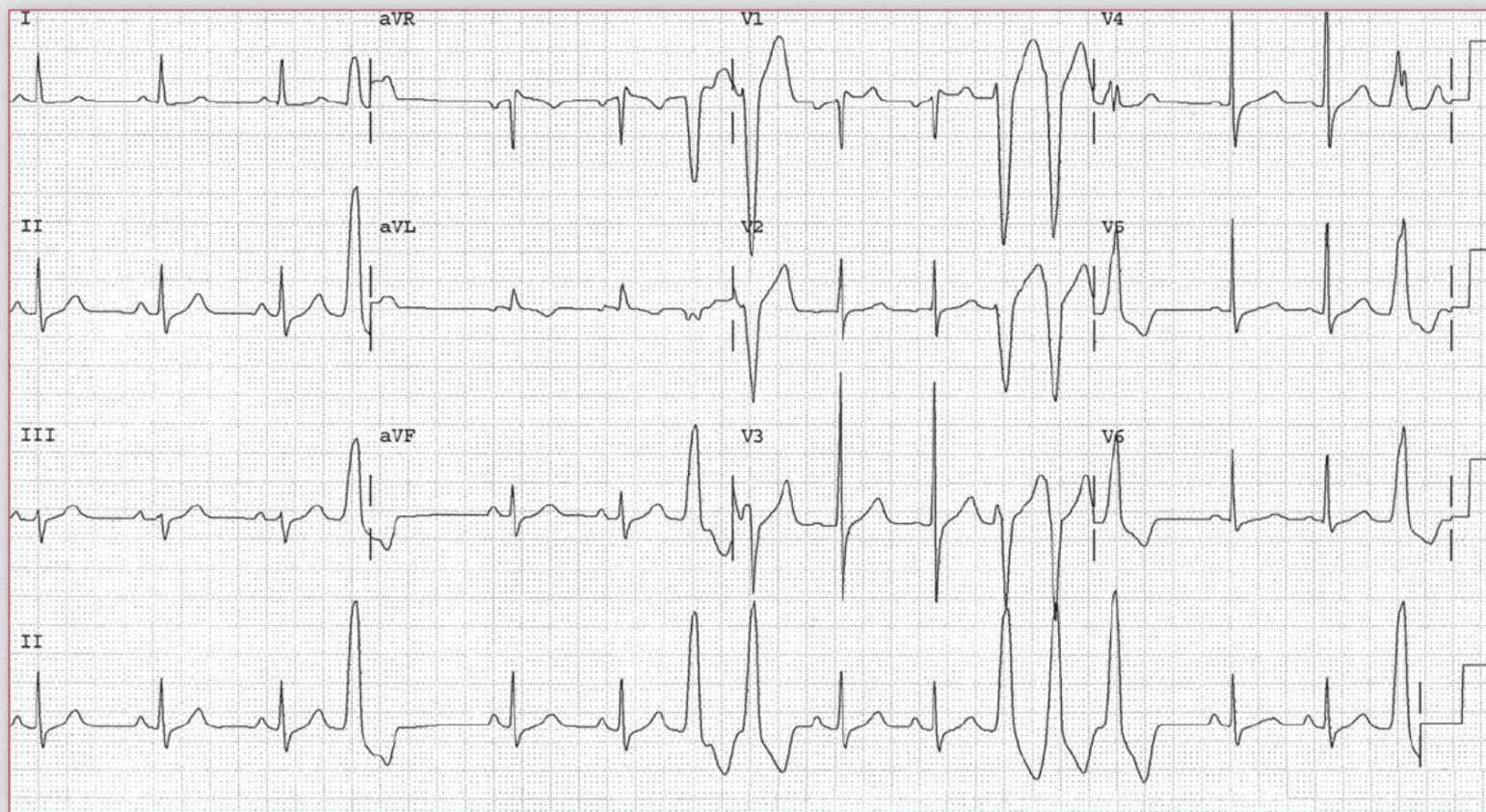
If a long rhythm strip is recorded, it can be observed that the interval between the PVCs is related to the underlying rate of the ectopic focus (*ie*, it is some integer of the underlying rate), and it may be possible to determine the actual rate of impulse activation from the focus. When the ectopic beats are infrequent, a very long rhythm strip is necessary to prove that there is an ectopic focus and a relationship between ectopic beats. The significance of PVCs due to a parasystolic focus is the same as with PVCs resulting from reentry. ■

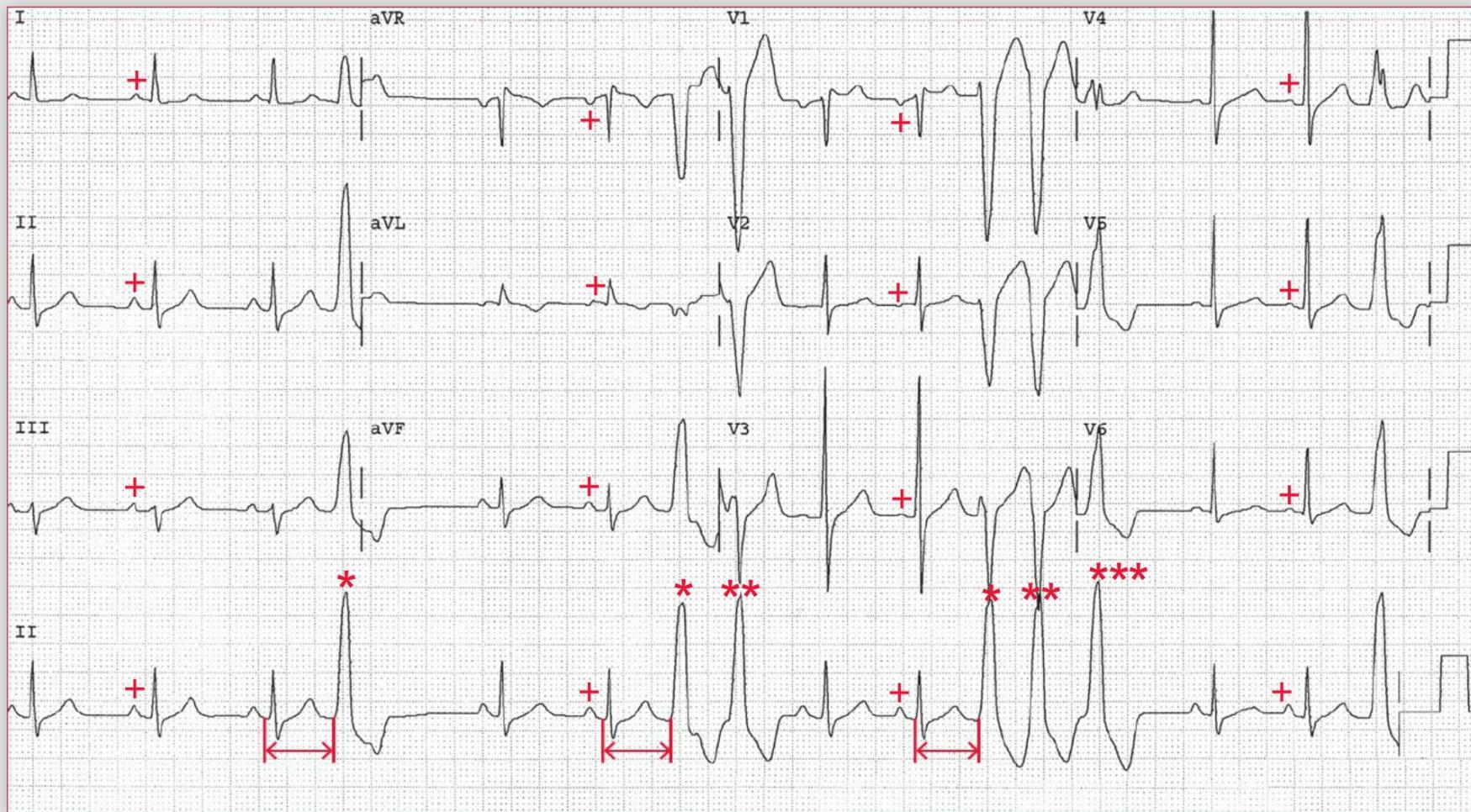
Notes

A 55-year-old man with no medical history presents to the emergency department because of palpitations that began several hours after attending a dinner party. During the party he had three cocktails, which is more than he usually drinks. He does not have any other associated symptoms. He describes the palpitations as forceful heartbeats rather than his heart racing. His physical examination is normal except for an irregular pulse rate and irregular heart sounds. An echocardiogram is obtained, showing normal right and left ventricular chamber size and normal contractility. An ECG is obtained.

What is the etiology of this patient's palpitations?

Are there any clinical implications?
Is therapy warranted?





ECG 47 Analysis: Normal sinus rhythm, unifocal premature ventricular complexes, ventricular couplets and ventricular triplets (nonsustained ventricular tachycardia)

The rhythm is regularly irregular, and there are narrow and wide QRS complexes. The irregularity is the result of early (premature) and wide QRS complexes (0.16 sec) that are not preceded by a P wave. Each of these QRS complexes has the same morphology, which is abnormal, not resembling a typical right or left bundle branch block. Hence they are unifocal premature ventricular complexes (PVCs). The first PVC is a single premature complex (*); the second occurrence of wide complexes includes two sequential ventricular complexes (*, **) called a ventricular couplet; and the third occurrence has three sequential ventricular complexes (*, **, ***). This is called a triplet or nonsustained ventricular tachycardia. Nonsustained ventricular tachycardia is defined as a series of three or more PVCs lasting up to 30 seconds or terminated earlier as a result of hemodynamic compromise. The coupling interval between the narrow complex and the PVC is fixed (↔), consistent with reentry as the mechanism for the arrhythmia.

The narrow QRS complexes (0.08 sec) are preceded by a P wave (+) with a fixed PR interval (0.18 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The underlying rate is 76 bpm. The QRS complex morphology is normal and the axis

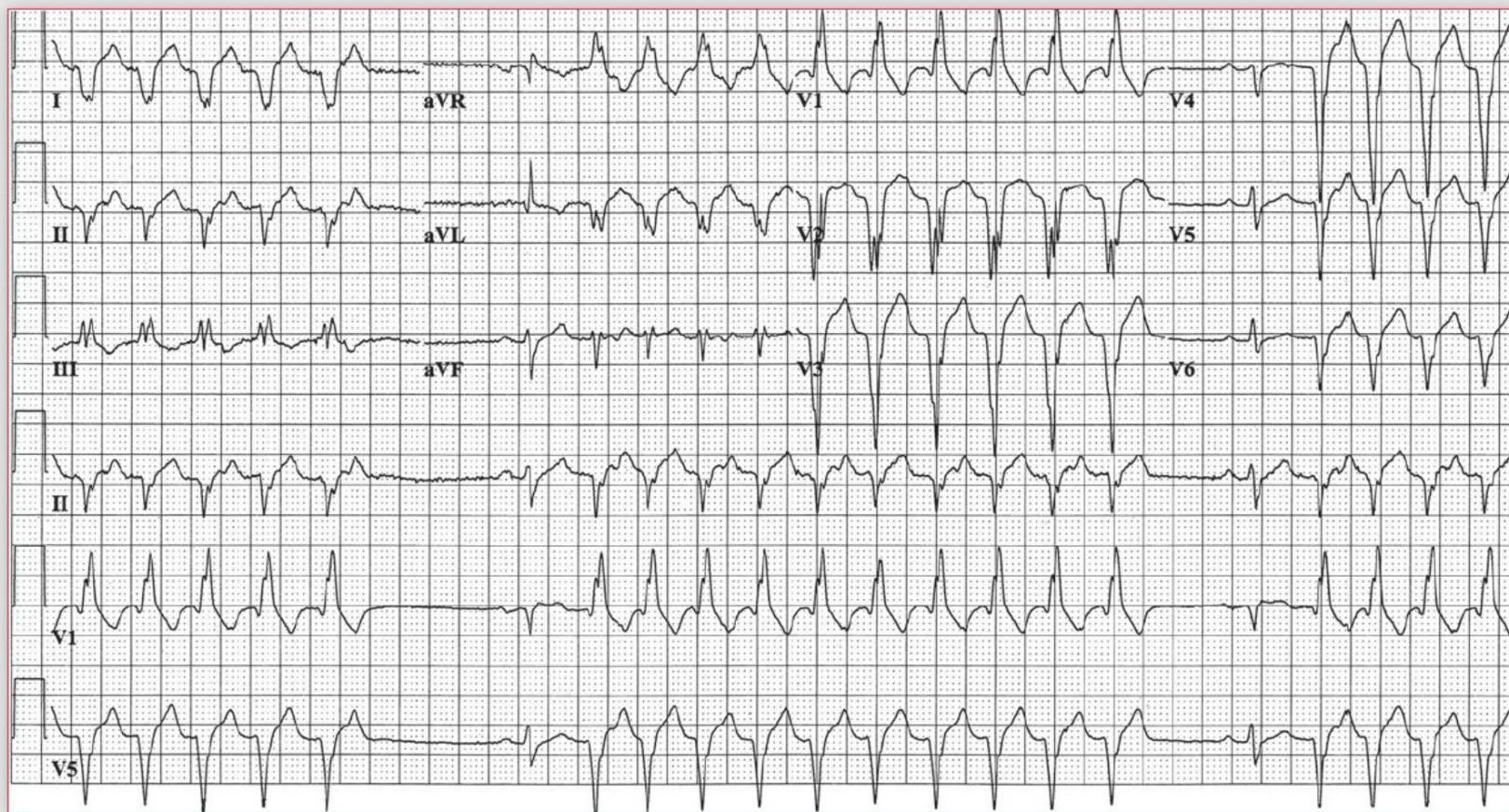
normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/430 msec).

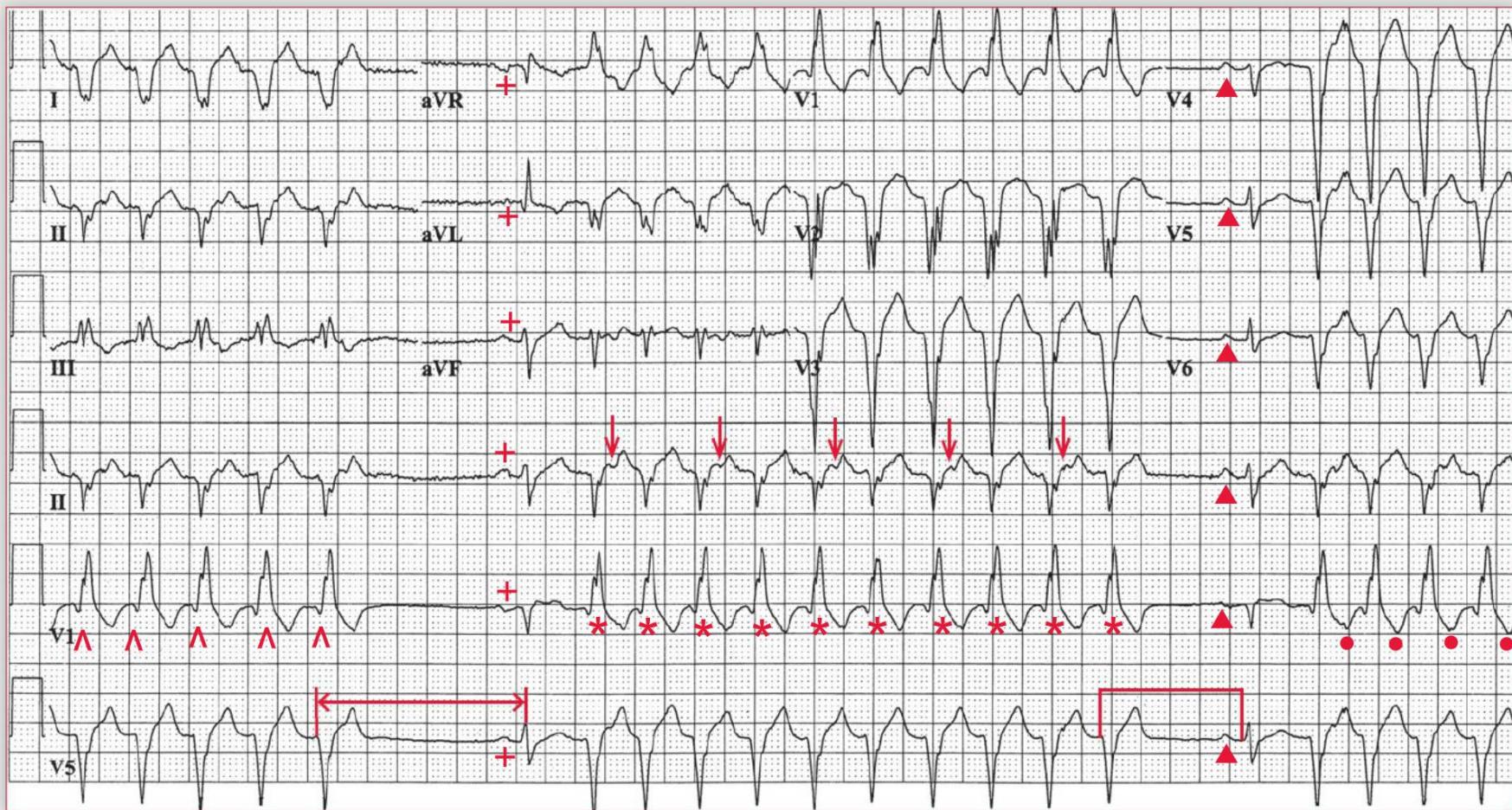
In the absence of structural heart disease, PVCs (including repetitive forms—couplets and triplets) are benign and not associated with an increased risk for ventricular tachyarrhythmia (ventricular tachycardia or ventricular fibrillation), which is the mechanism for sudden cardiac arrest. It is likely that the arrhythmia was induced by excessive alcohol consumption, although this relationship has not been well established. There is no indication for therapy unless the ventricular arrhythmia is associated with disturbing side effects. This patient's symptoms of palpitations are not serious and may very well be transient. The palpitations are usually the result of post-extrasystolic potentiation that follows the PVC. This is due to the larger volume of blood in the left ventricle as a result of the pause and the Frank-Starling effect, which results in an increase in inotropy. ■

Notes

A 71-year-old woman with ischemic cardiomyopathy (ejection fraction 32% by echocardiography) is admitted with symptoms of pre-syncope. She is noted on ECG to have runs of wide complex tachycardia during which she feels slightly faint. Physical examination reveals an irregularity in her heart rhythm, during which her pulse is barely palpable.

What is the likely etiology of her pre-syncope?
What therapies are indicated?





ECG 48 Analysis: Normal sinus rhythm, nonsustained monomorphic ventricular tachycardia with 2:1 retrograde conduction

There are runs of a tachycardia (rate 140 bpm) with wide QRS complexes (0.14 sec). All of the QRS complexes have a similar morphology (▲), which is abnormal and does not resemble a typical right or left bundle branch block. The first run terminates abruptly, and after a pause of 2.8 seconds (↔) there is a narrow QRS complex (0.08 sec) with a P wave (+) preceding it and a PR interval of 0.18 second. The P wave is positive in leads II and V5 and is likely sinus. The QRS complex is negative in leads II and aVF and, although not seen in lead I, it is likely an extreme left axis, between -30° and -90° (*ie*, a left anterior fascicular block).

Immediately following the narrow QRS complex there is a second run (10 beats) of the same wide complex tachycardia at a rate of 140 bpm (*); the QRS complex morphology in this run is identical to that in the first run. This run again abruptly terminates and, after a 1.92-second pause (▀), there is a narrow QRS complex with the same morphology as was seen before. This narrow QRS complex is preceded by a P wave (▲) and PR interval that are the same as those associated with the first narrow QRS complex. This is, therefore, another sinus complex. A third instance of the wide complex tachycardia (●) is again present.

There are no P waves before any of the wide QRS complexes of the tachycardia. However, it can be seen that there is a notching of the ST segment in every other QRS complex in leads II, aVF, and V1, particularly in lead II (↓). As the ST segment should be smooth, notching suggests a superimposed P wave. Although it is not definite, these are probably retrograde P waves. Hence there is 2:1 retrograde conduction or ventriculoatrial (VA) block. In addition, the axis of the wide

QRS complexes is indeterminate, between $+90^{\circ}$ and $+/-180^{\circ}$ (negative QRS complex in leads I and aVF), as a result of either an extreme left or right axis. An indeterminate axis associated with a wide QRS complex is due to direct myocardial activation as occurs with a ventricular complex, a preexcited complex (Wolff-Parkinson-White), or a paced complex (primarily with biventricular pacing). The wide QRS complexes have an abnormal morphology, without evidence of preexcitation or pacing. Hence they are ventricular in origin and, therefore, these are runs of nonsustained monomorphic ventricular tachycardia that self-terminate.

There are several diagnostic criteria for ventricular tachycardia:

- QRS complexes that are wide (> 0.12 sec) and abnormal in morphology not resembling either a typical left or right bundle branch block.
- The presence of P waves that are dissociated from QRS complexes (AV dissociation, *ie*, variable PR intervals) with a ventricular rate that exceeds the atrial rate. A negative P wave may be seen after the QRS complex if VA conduction is present. This may also be seen with supraventricular tachycardia. However, the presence of intermittent retrograde VA block is consistent with ventricular tachycardia. Often no P waves are seen. The presence of AV dissociation or any retrograde VA block with a wide complex tachycardia is most important for establishing ventricular tachycardia as the etiology. Supraventricular tachycardias are generally not dissociated, nor is intermittent retrograde block seen.

continues

- QRS complexes and ST-T waves that show non-rate-related variability in morphology. This variability is not seen with any supraventricular tachyarrhythmia because, regardless of the etiology of the arrhythmia (sinus, atrial or AV nodal, or junctional), the pathway from atrium to ventricle is fixed (*ie*, the normal His-Purkinje system) and is always the same from beat to beat. In contrast, ventricular tachycardia is due to a reentrant circuit located within the ventricular muscle that bypasses the normal His-Purkinje activation pathway. This may result in changes in the circuit or in the direction of ventricular activation and changes in repolarization, producing changes in QRS complex morphology and ST-T waves. The ST-T wave changes may also result from superimposed P waves.
- Fusion or captured (Dressler) complexes. These are features associated with AV dissociation. Intermittent AV conduction with fusion complexes and Dressler complexes (intermittent captured complexes) is seen with AV dissociation and is most often present when the ventricular rate is slower, allowing more time for antegrade conduction through the AV node. The impulse resulting from antegrade conduction through the AV node and His-Purkinje system may join with the impulse generated by the ventricular focus, resulting in a fusion complex (which has a morphology resembling both the supraventricular as well as the ventricular complex) or a completely captured complex or Dressler complex (which is due to complete capture of the ventricular myocardium by the impulse conducted via the normal conduction system).

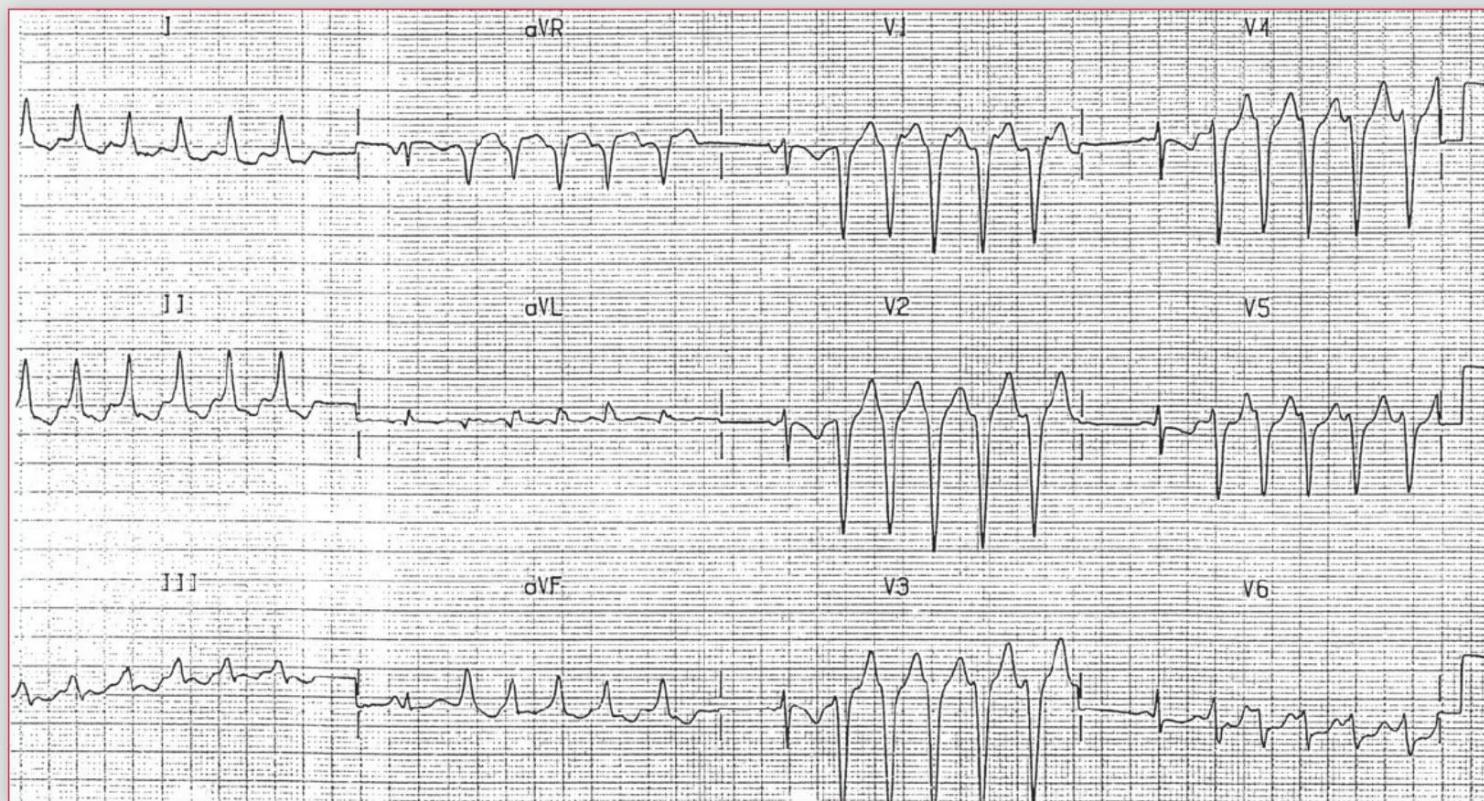
- An indeterminate axis, between -90° and $+/-180^\circ$ (*ie*, negative QRS complex in leads I and aVF). An indeterminate axis with a wide QRS complex rhythm is the result of any impulse that directly activates the left ventricular myocardium (*ie*, ventricular tachycardia, Wolff-Parkinson-White pattern, or biventricular paced complex).
- Positive QRS complex concordance (*ie*, a tall R wave in leads V1-V6), which is seen with any impulse that directly activates the left ventricular myocardium (*ie*, ventricular tachycardia or Wolff-Parkinson-White pattern). Negative QRS complex concordance is not as useful because this pattern may be seen with a left bundle branch block.

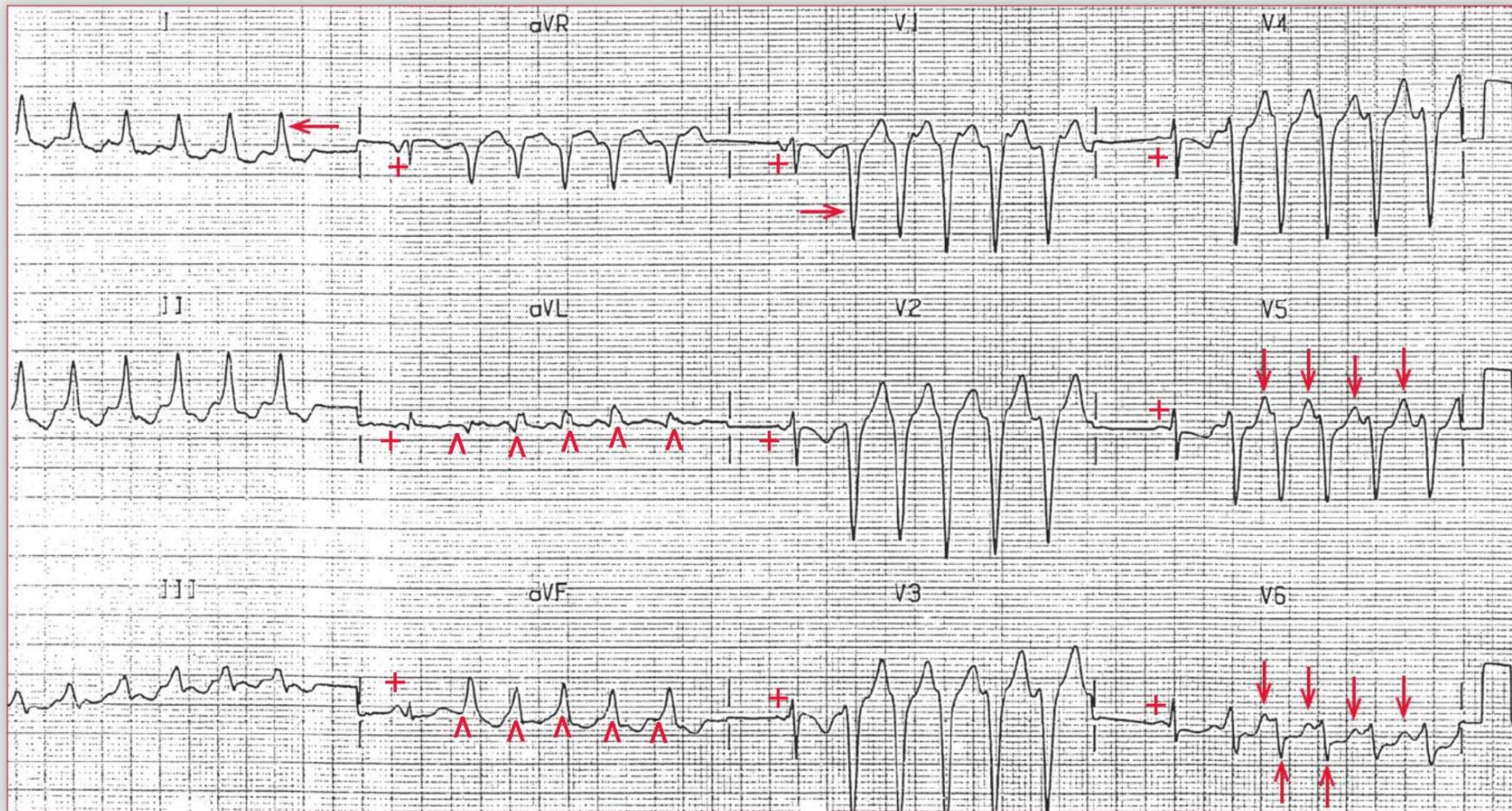
In the context of cardiomyopathy, ventricular tachycardia is often not well tolerated hemodynamically. In this case, the patient's pre-syncope was likely caused by ventricular tachycardia and associated hypotension. Given the symptomatic nonsustained ventricular tachycardia, suppressive anti-arrhythmic therapy for symptom relief is warranted. In patients with ischemic cardiomyopathy who have nonsustained ventricular tachycardia there is a high risk for sustained ventricular tachyarrhythmia (ventricular tachycardia or ventricular fibrillation). Hence, these patients would also be candidates for an implantable cardioverter-defibrillator to prevent sudden cardiac arrest as a result of a sustained life-threatening ventricular tachycardia. ■

A 24-year-old woman with no history of heart disease is referred to a cardiologist after experiencing sustained palpitations while training for a marathon. She states that she has had a long history of brief palpitations, but this was the first time they lasted more than a few minutes. She had gone to a local emergency department, but the palpitations had resolved by the time she arrived. Her evaluation at that time was unremarkable,

as was an ECG. Her cardiology evaluation includes a full examination, which is normal. An echocardiogram is normal and shows no evidence of valvular or other structural heart disease. Left ventricular ejection fraction is 60%. She is scheduled for Holter monitoring, but 1 week later she again notes episodic palpitations for which she returns to the emergency department, where the following ECG is obtained.

**What does this ECG show?
What is the etiology for the arrhythmia noted?**





ECG 49 Analysis: Normal sinus rhythm, nonsustained ventricular tachycardia, right ventricular outflow tract tachycardia (repetitive monomorphic ventricular tachycardia)

There is a regularly irregular rhythm with runs of both wide and narrow QRS complexes. The runs of wide QRS complexes are occurring in a bigeminal pattern. Noted are several narrow QRS complexes with a duration of 0.08 second. These complexes are preceded by a P wave (+) with a stable PR interval (0.14 sec). The P wave is positive in leads aVF and V4-V6. Although there is no narrow complex seen in leads I, II, or III, these are probably normal sinus complexes. After each sinus complex there are five sequential wide QRS complexes that have a duration of 0.14 second and a rate of 190 bpm. The QRS complexes have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The complexes have a morphology that resembles a left bundle branch block (LBBB), with a broad R wave (←) in lead I and a QS complex in lead V1 (→). However, there is a terminal S wave in lead V6 (↑), which is atypical for an LBBB. The QRS complexes show variability in morphology (Λ), particularly evident in leads aVL and

aVF. In addition, there are subtle differences in ST-T wave morphology (↓), especially evident in leads aVF and V5-V6. Therefore, the wide QRS complexes are ventricular and these are episodes of nonsustained ventricular tachycardia (NSVT). NSVT has been defined as three or more sequential ventricular complexes lasting up to 30 seconds or terminated earlier because of hemodynamic compromise.

The QRS complex axis, which is inferiorly directed or normal, and morphology (resembling an LBBB and consistent with a right ventricular origin) are characteristic of ventricular complexes originating from the right ventricular outflow tract (RVOT); hence this is RVOT tachycardia. This presentation, with episodes of NSVT, is termed repetitive monomorphic ventricular tachycardia. In the presence of catecholamines or sympathetic drive, as occurs with exercise, the arrhythmia may become sustained, and hence typical RVOT tachycardia.

continues

Repetitive monomorphic ventricular tachycardia is characterized by frequent short “salvos” or runs of monomorphic NSVT. It has been variously termed right ventricular tachycardia, RVOT tachycardia, catecholamine-sensitive ventricular tachycardia, adenosine-sensitive ventricular tachycardia, and exercise-induced ventricular tachycardia. It occurs almost exclusively in young to middle-aged individuals who usually do not have any structural heart disease. There has generally

been no predilection on the basis of gender. Most arrhythmias are nonsustained (usually three to 15 beats), but up to 50% of patients have some sustained episodes, which are generally provoked by sympathetic stimulation or elevated catecholamine levels. Some patients have only sustained ventricular tachycardia. Episodes of NSVT as well as sustained ventricular tachycardia are typically provoked by emotional stress or exercise. Right ventricular tachycardias usually originate from the septal aspect of the RVOT. The QRS complex morphology has a characteristic appearance with two main features: LBBB and an inferior (or normal) axis.

This arrhythmia cannot usually be induced in the electrophysiology laboratory with programmed stimulation. In most patients, sustained or nonsustained episodes occur in response to burst atrial or ventricular pacing and are greatly facilitated by isoproterenol or epinephrine infusions. These observations suggest that the mechanism is more likely triggered activity due to delayed after-potentials or possibly an ectopic ventricular focus rather than reentry.

This arrhythmia is associated with a good prognosis, and the incidence of hemodynamic compromise or sudden cardiac death is very low. Treatment includes a β -blocker, verapamil, a standard anti-arrhythmic drug, or ablation. Ablation has become the treatment of choice as the majority of patients are young and drug therapy is often not preferred. ■

Core Case 50

A 57-year-old man presents to the emergency department with a several-hour history of substernal chest burning radiating to his throat and left arm associated with diaphoresis and nausea. An ECG (50A) is obtained. He is then placed on telemetry and given intravenous heparin and nitroglycerin, with

ECG 50A



improvement in the chest discomfort. As a result of changes on the ECG a cardiology consult is called. While waiting, the man develops a rapid heart rate with loss of consciousness (ECGs 50B and 50C). CPR is begun, and he is successfully resuscitated. He is quickly brought to the cardiac catheterization laboratory.

ECG 50B



Core Case 50

ECG 50C

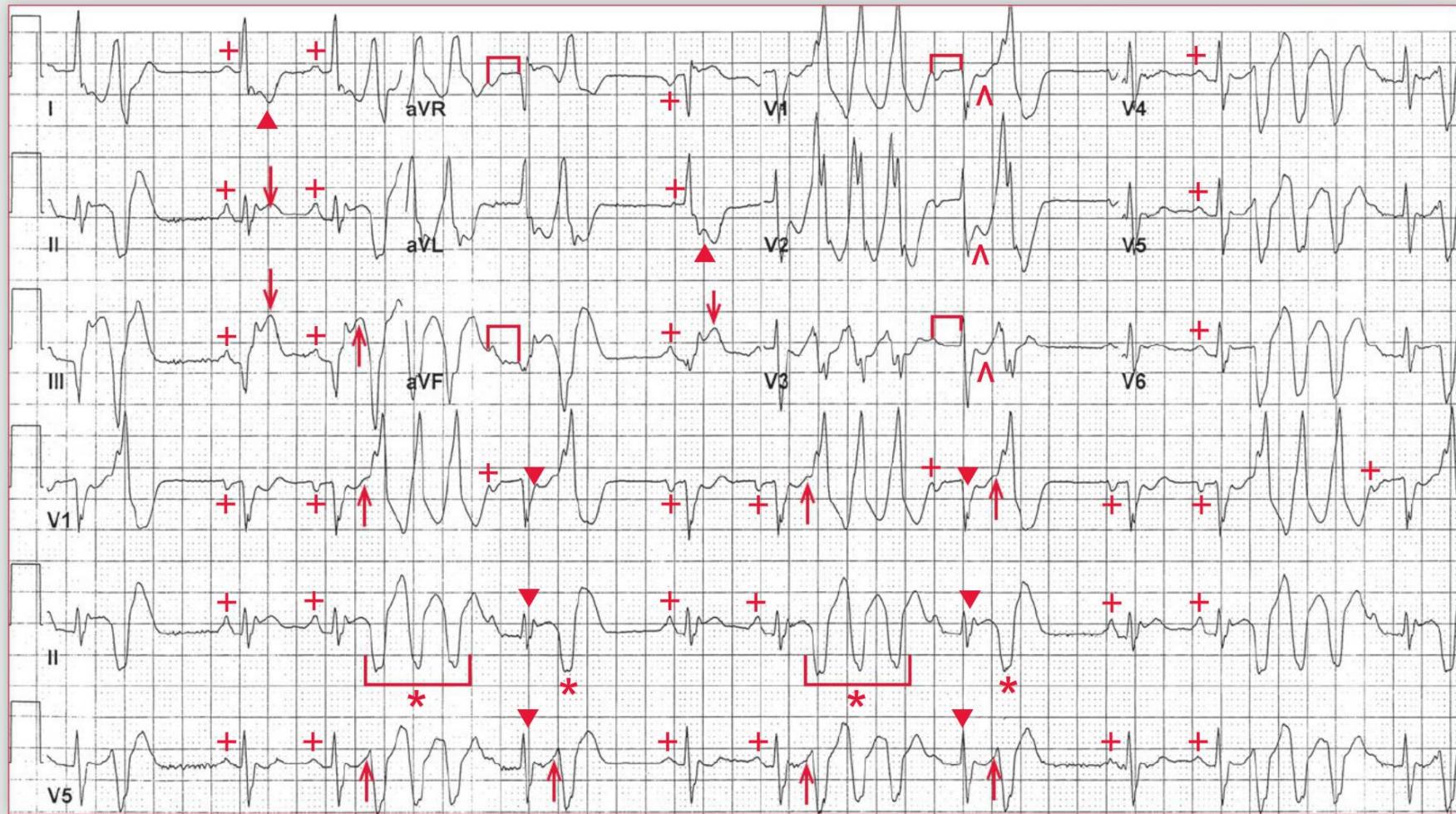


What abnormalities are notable in ECG 50A?

What is the diagnosis?

What does the telemetry monitoring (ECGs 50B and 50C) show?

What is the etiology of the abnormalities noted?



ECG 50A Analysis: Sinus tachycardia, left axis, acute inferior wall myocardial infarction, unifocal premature ventricular complexes (R on T), monomorphic nonsustained ventricular tachycardia, retrograde concealed conduction

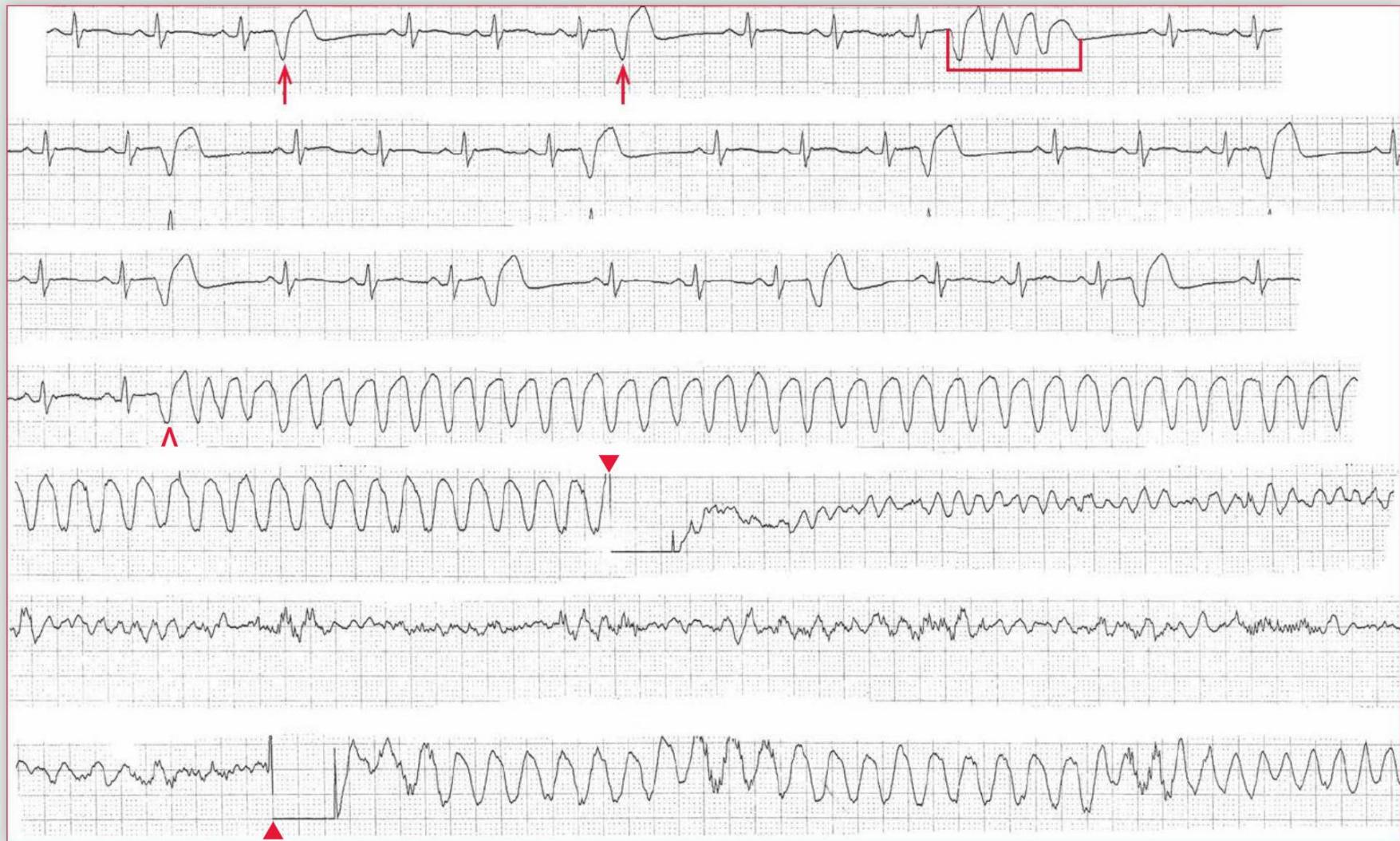
ECG 50A shows a regular rhythm at a rate of 100 bpm. There is a P wave (+) before each of the narrow QRS complexes with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6). Therefore, this is sinus tachycardia. These QRS complexes have a normal duration (0.08 sec) and a left axis, between 0° and –30°, with a positive QRS complex in leads I and II and a negative QRS complex in lead aVF. The QT/QTc intervals are normal (290/440 msec). There is ST-segment elevation (↓) noted in leads II, III, and aVF, indicating an acute inferior wall myocardial infarction. There is ST-segment depression (▲) in leads I and aVL (which are reciprocal changes) and also in leads V1-V3 (Δ), which may be reciprocal changes or represent posterior wall involvement.

Also seen are wide QRS complexes (*) (0.16 sec) that are premature and are not preceded by a P wave. They have an abnormal morphology and do not resemble a typical right or left bundle branch block. These are ventricular in origin, and there are single premature ventricular complexes (PVCs) as well as triplets (□) (also termed nonsustained ventricular tachycardia [NSVT]). Each of the PVCs has the same morphology, and hence they are unifocal. Additionally, each QRS complex of the triplet has the same morphology; hence this is monomorphic NSVT. After each triplet, there is a sinus complex (▼), but the PR interval is longer (□) (0.26 sec) than the baseline PR interval (0.16 sec). This is a result of retrograde concealed conduction; that is,

the ventricular complex preceding this sinus complex results in partial retrograde conduction into the AV node. However, the impulse does not conduct completely through the node (*ie*, it is concealed within the AV node), resulting in partial AV nodal depolarization and partial prolongation in its refractoriness. Hence the next atrial waveform gets through the AV node but with a longer conduction time.

Of importance is the fact that the PVCs are very early and are indeed occurring just after the peak of the T wave (↑). These are termed R-on-T PVCs. This part of the T wave corresponds to the vulnerable period of the fast action potential and is the time immediately after the end of phase 3 and the beginning of phase 4 when the membrane potential transiently becomes more negative (hyperpolarized) than the resting membrane potential of –90 mV. It is at this time during the action potential when an electrical stimulus of enough energy can provoke ventricular flutter, polymorphic ventricular tachycardia, or ventricular fibrillation. The amount of energy necessary is termed the ventricular fibrillation threshold. In the normal myocardium the ventricular fibrillation threshold is high and PVCs do not generate enough energy to provoke arrhythmia. However, ischemia lowers the ventricular fibrillation threshold and in this situation the PVCs generate enough electrical energy to provoke ventricular arrhythmia. Hence the R-on-T PVCs are serious and can result in serious ventricular tachyarrhythmias in the ischemic myocardium.

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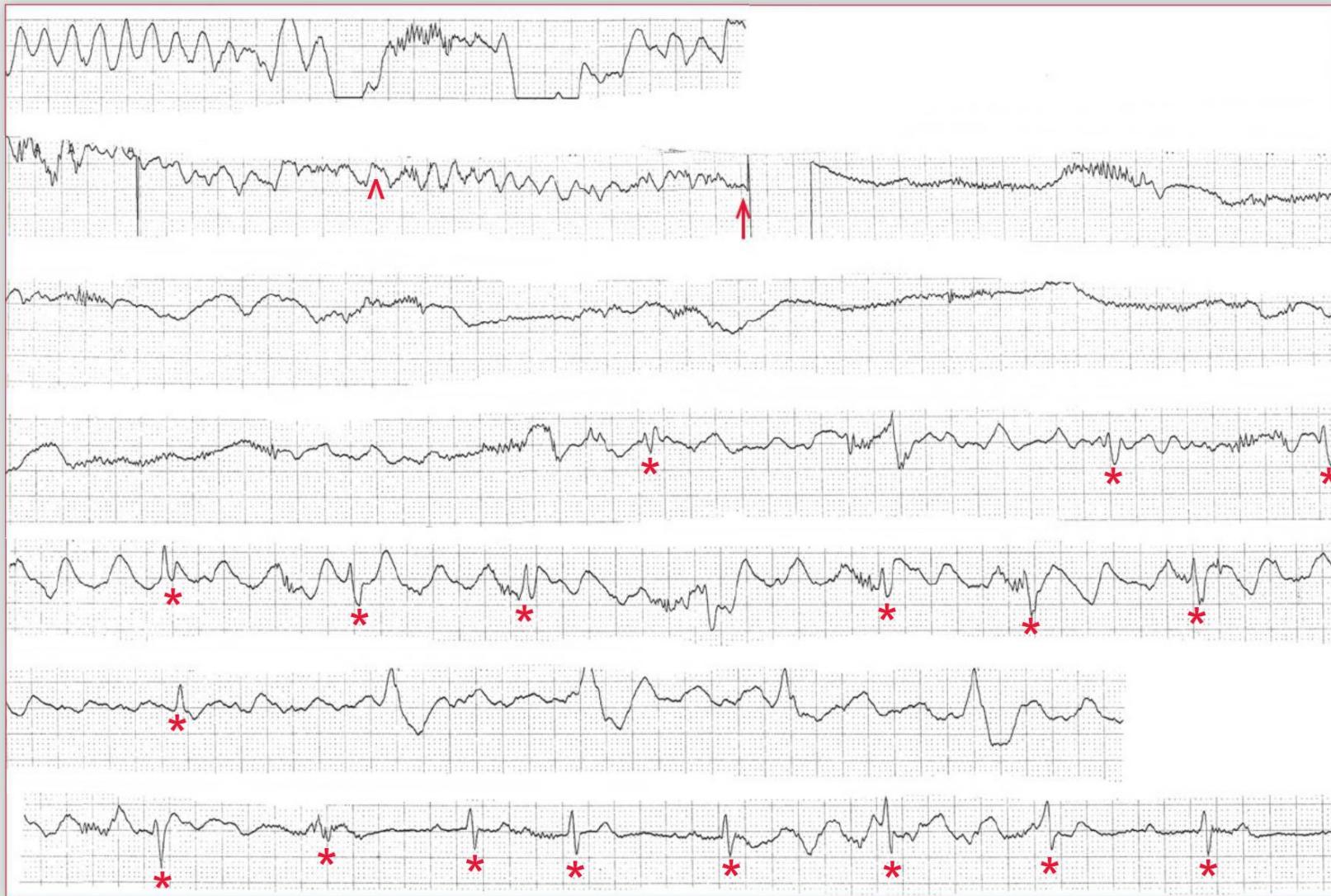


ECG 50B Analysis: Sustained monomorphic ventricular tachycardia
(ventricular flutter), ventricular fibrillation, polymorphic ventricular tachycardia

ECG 50B shows continuous telemetry monitoring strips. The first strip shows normal sinus rhythm with two R-on-T PVCs (↑) and a single four-beat episode of NSVT (□). On the fourth strip the R-on-T PVC (Λ) results in an episode of sustained monomorphic ventricular tachycardia with a rate exceeding 260 bpm; this is often called ventricular flutter. On the fifth strip there is a low-energy synchronized shock from a cardioverter (▼). However, as it is impossible to distinguish the QRS complex from the T wave, cardioversion, which is the delivery of a synchronized shock during the QRS complex, is not appropriate as the machine is not likely to distinguish between the QRS complex and the T wave. Therefore, there is a 50% chance of delivering a low-energy shock on the T wave, which would have a high probability of provoking

rapid ventricular tachycardia or ventricular fibrillation. Thus when it is hard to distinguish the QRS complex from the T wave, high-energy defibrillation (nonsynchronized delivery of energy) should be used in order to avoid the delivery of a low-energy shock on the T wave. With high-energy defibrillation the entire heart is defibrillated and made refractory, allowing for the restoration of a sinus mechanism. In this case (as can be seen on the fifth strip) it appears that the shock was delivered during the T wave (▼) and immediately after the delivery of the shock ventricular fibrillation was provoked (strips 5 and 6). A second defibrillation (nonsynchronized) shock (▲) (seen on strip 7) reverted the ventricular fibrillation to polymorphic ventricular tachycardia, as can be seen at the end of strip 7.

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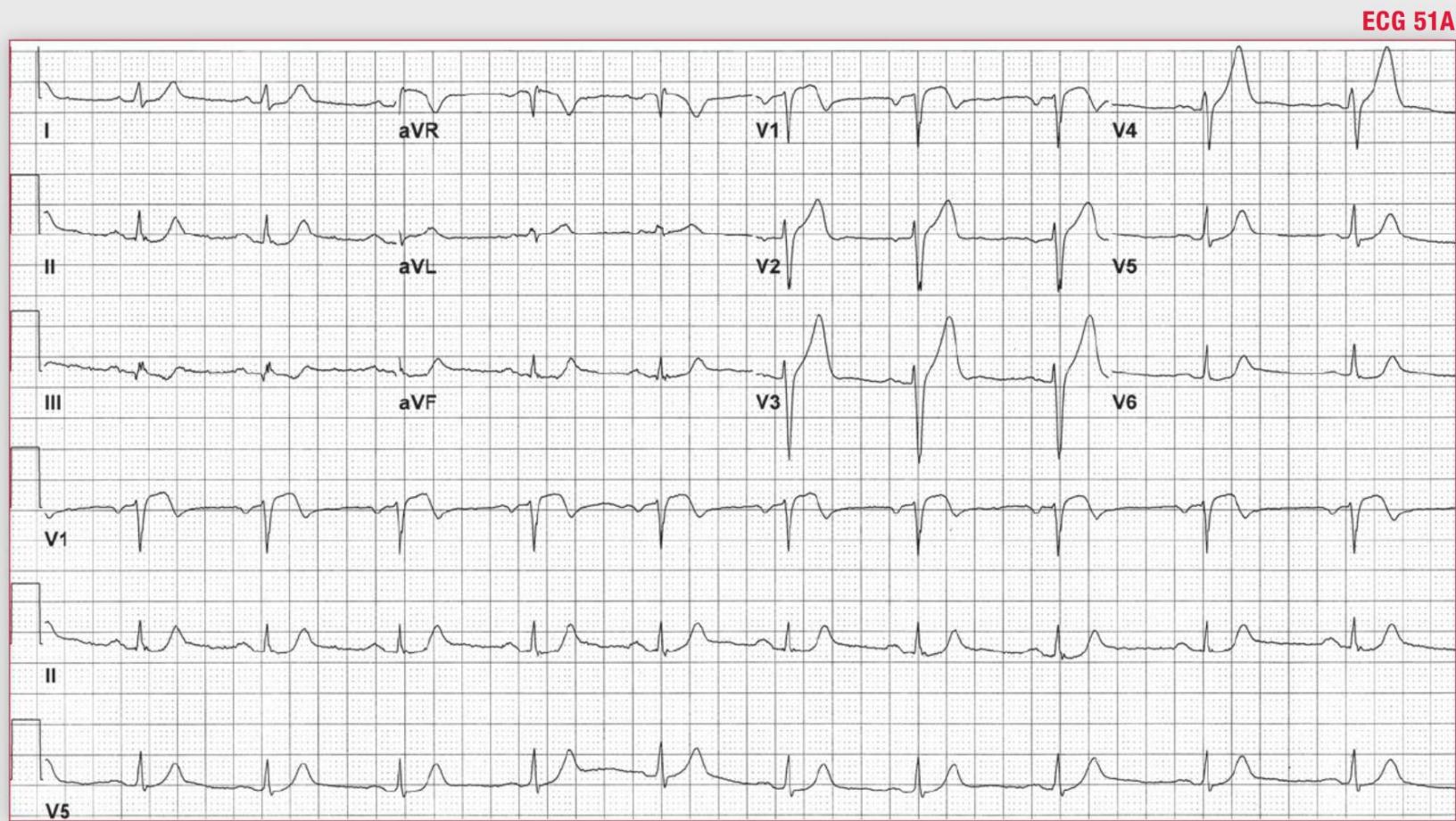


ECG 50C Analysis: Ventricular fibrillation, sinus rhythm

ECG 50C is a continuation of the telemetry monitoring strips seen in ECG 50B. On the first strip the polymorphic ventricular tachycardia degenerates into ventricular fibrillation (\wedge) (strip 2), which is successfully reverted with defibrillation (\uparrow). This is followed by a long period of asystole (end of strips 2 and 3), after which organized QRS complexes (*) can be seen in strips 4 to 7 (in addition to artifact from chest compressions). ■

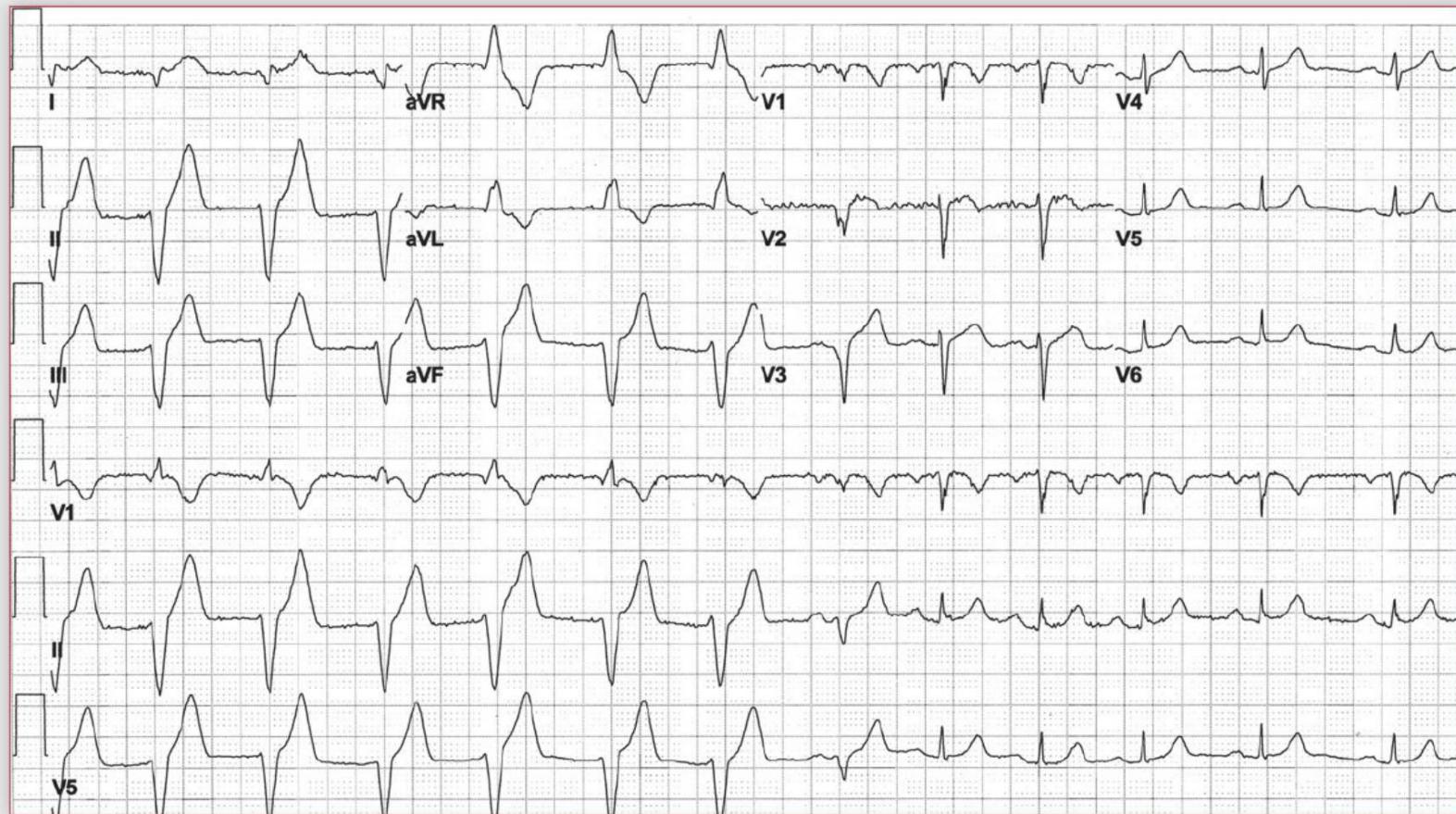
Core Case 51

A 63-year-old woman presents with the acute onset of chest discomfort, and an ECG (51A) is obtained. Her initial troponin I level is slightly elevated, and she is



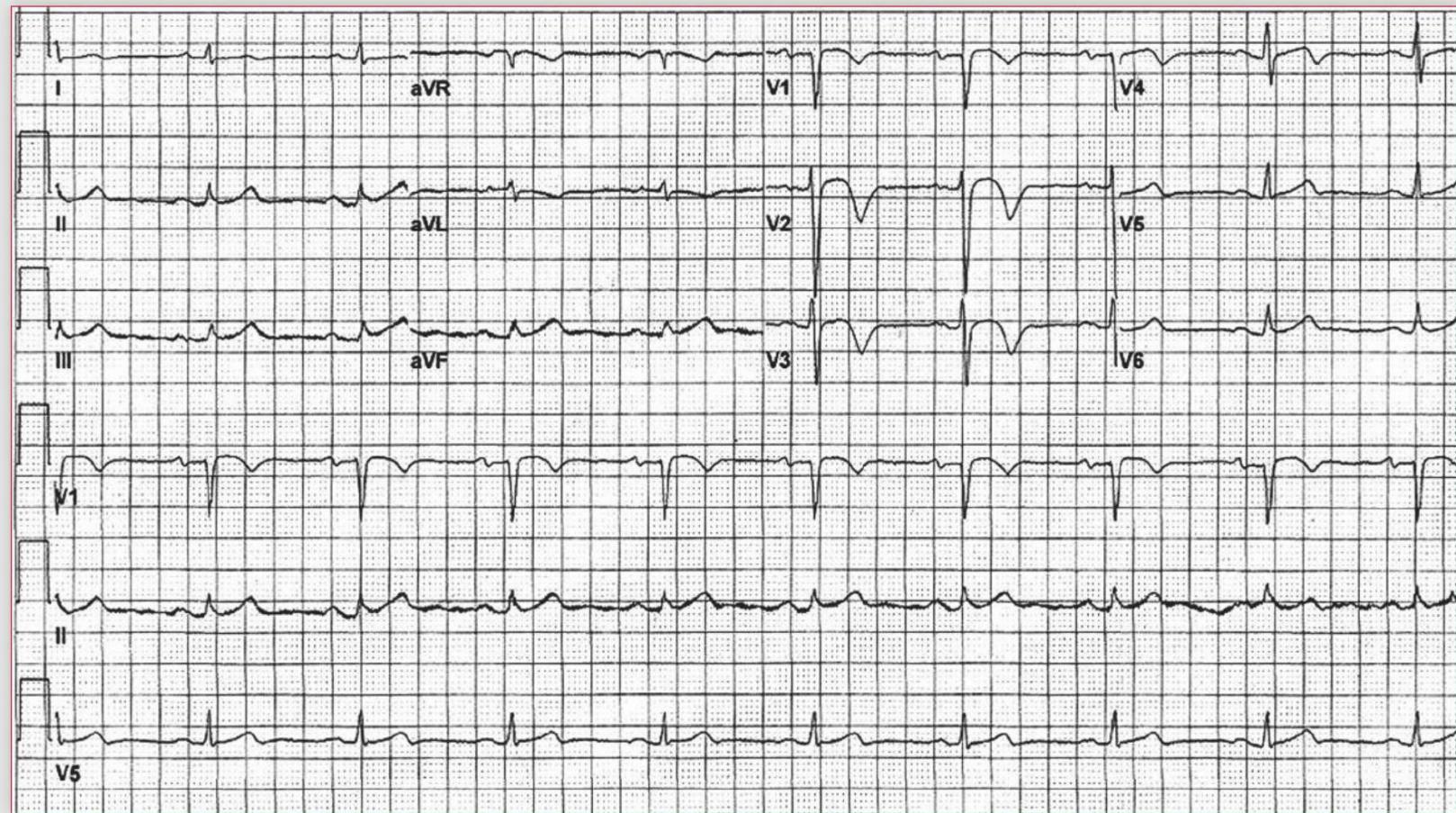
taken for urgent cardiac catheterization. Immediately after the procedure, a second ECG (51B) is obtained. A third ECG (51C) is obtained the next day.

ECG 51B



Core Case 51

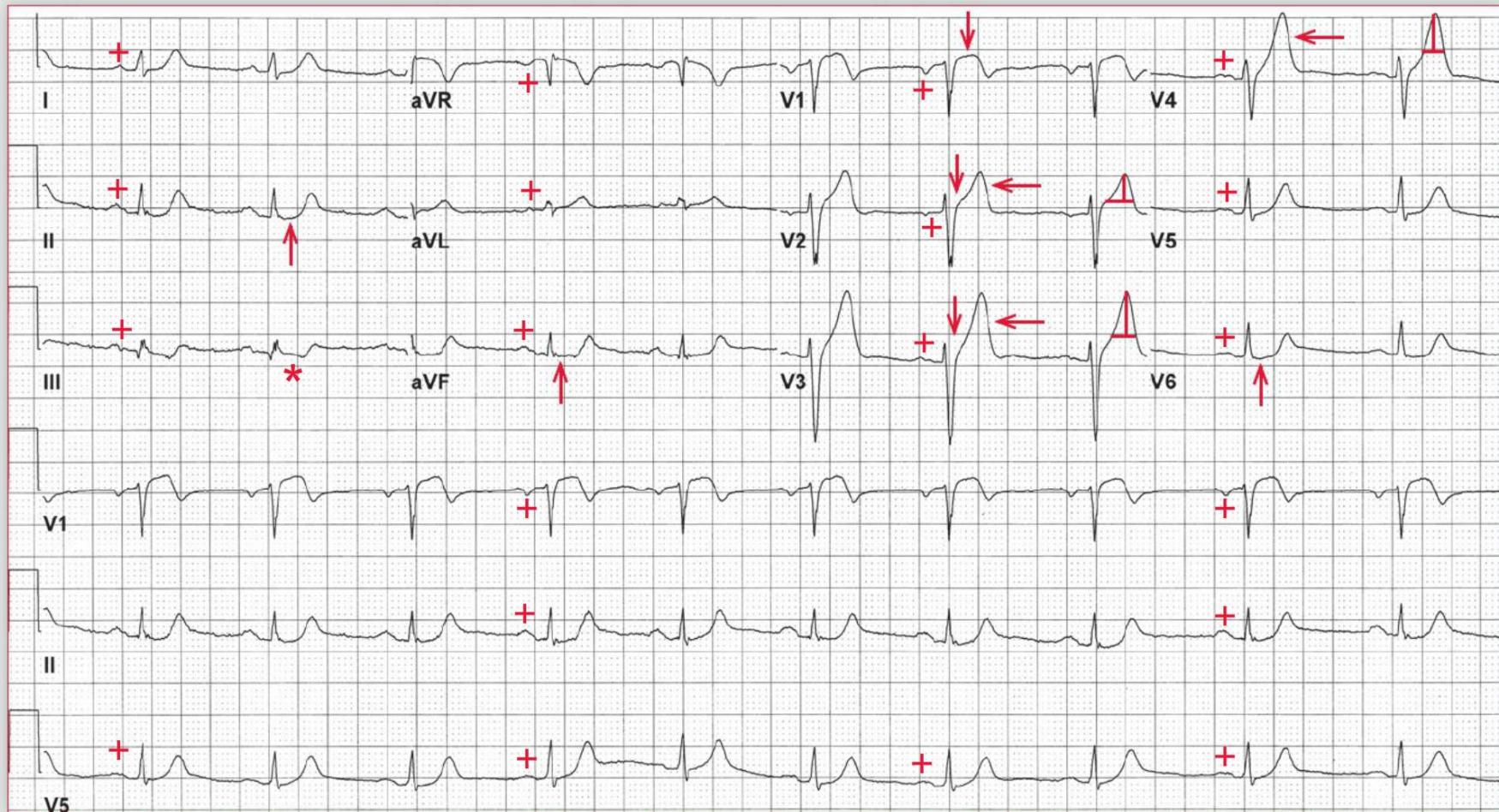
ECG 51C



What abnormality can be seen in ECG 51A?

What is the rhythm abnormality in ECG 51B?

How would you manage this rhythm disturbance?

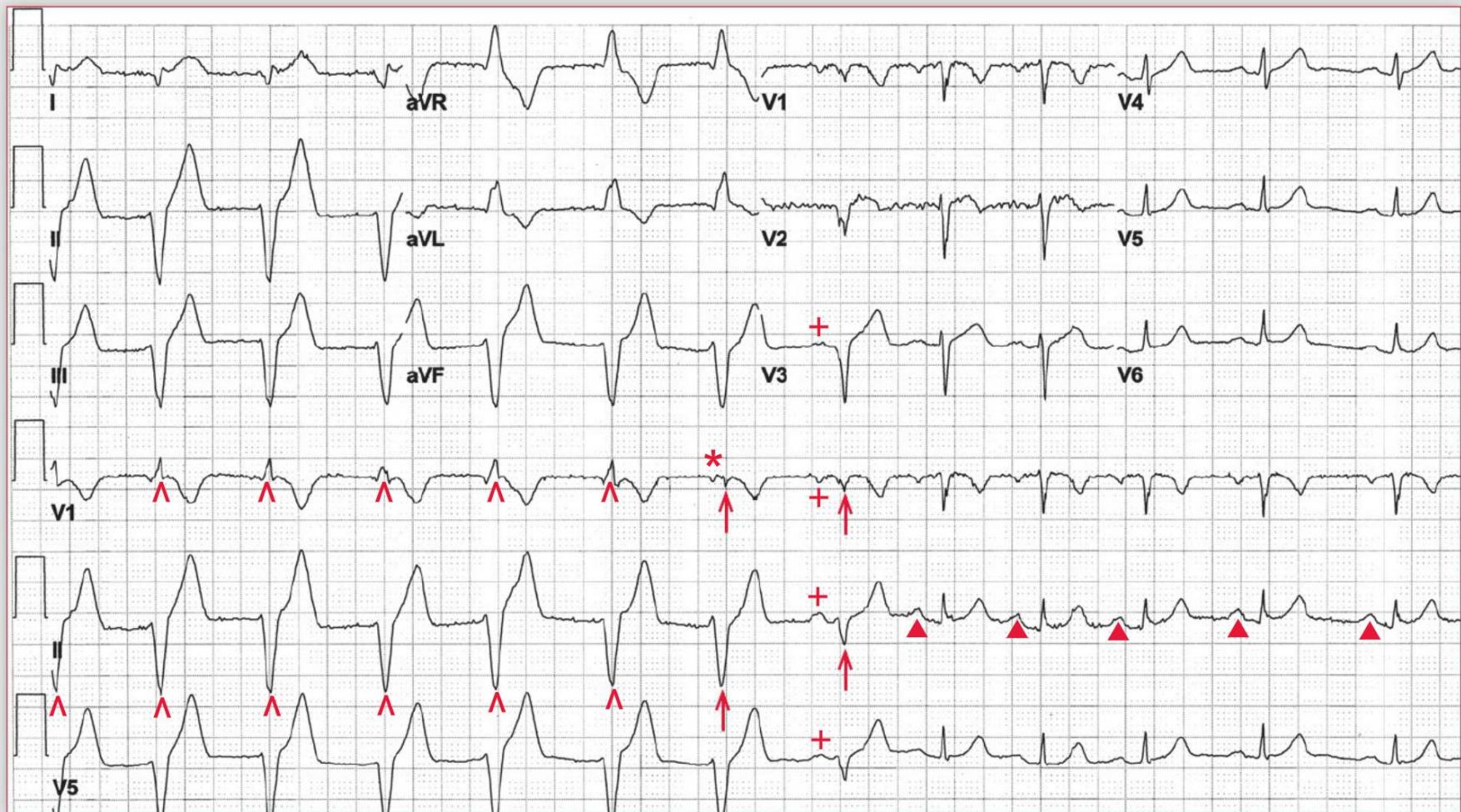


ECG 51A Analysis: Normal sinus rhythm, acute anterior myocardial infarction
(slight ST-segment elevation and hyperacute T waves)

ECG 51A shows a regular rhythm at a rate of 64 bpm. There is a P wave (+) before each QRS complex with a constant PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. This is, therefore, a normal sinus rhythm. The QRS complex duration (0.08 sec) is normal and it has a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/390 msec). Noted is ST-segment elevation in leads V1-V3 (↓) and slight ST-segment depression in leads II, III, aVF, and V6 (↑). In addition, the T waves in leads V1-V4 (←) are tall and symmetric. This ECG is consistent with a very early acute anterior wall ST-segment elevation myocardial infarction (STEMI). The ST-segment depressions in leads II, III, aVF, and V6 are reciprocal changes.

The presence of STEMI is confirmed by the slightly elevated troponin I levels. Patients with STEMI require urgent revascularization, preferably with angioplasty and stenting. If not immediately available, a thrombolytic agent is indicated. This patient did undergo cardiac catheterization, which revealed an acute thrombotic occlusion of the left anterior descending artery.

continues



ECG 51B Analysis: Accelerated idioventricular rhythm,
fusion complexes, sinus rhythm with first-degree AV block

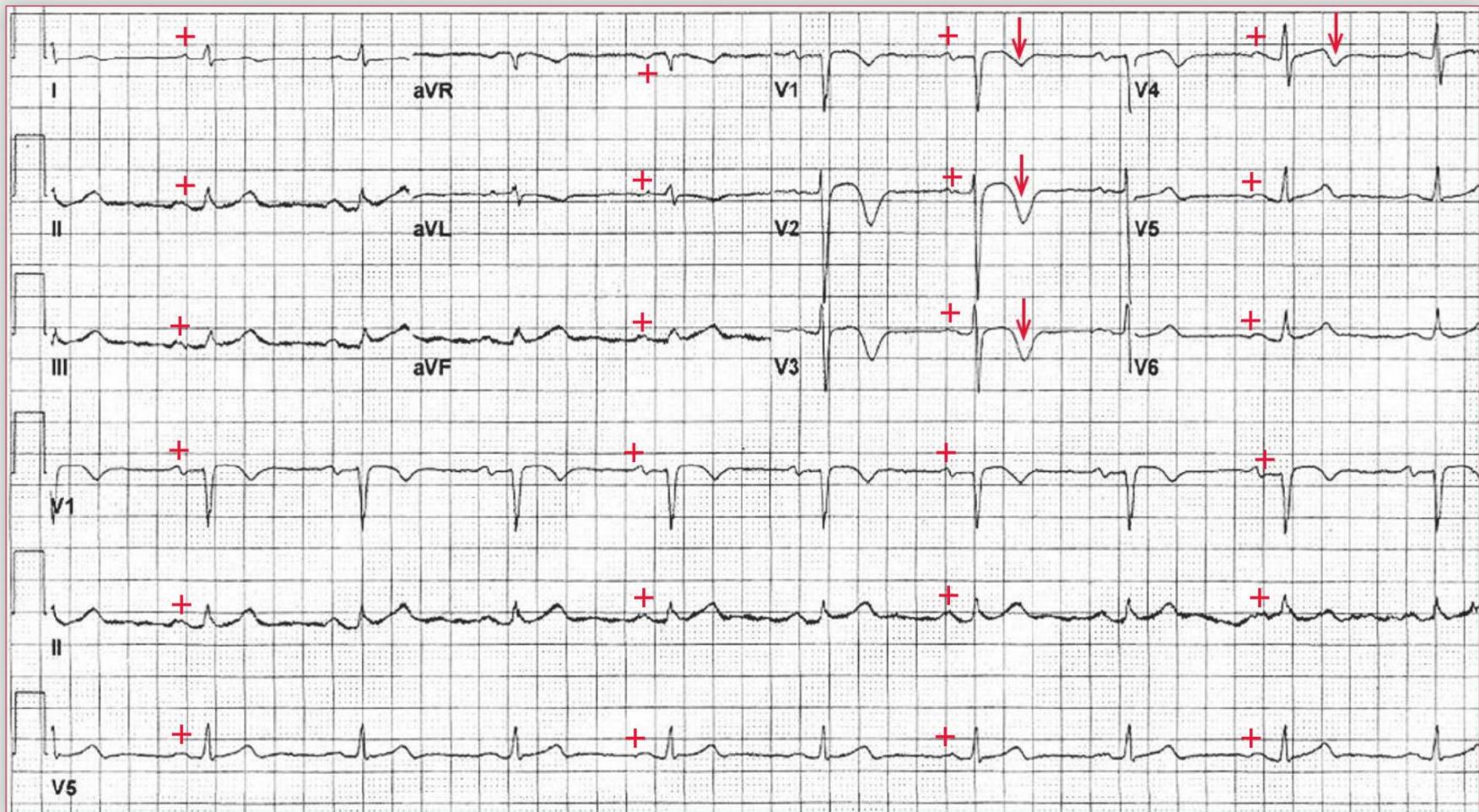
ECG 51B was obtained shortly after the left anterior descending artery was opened and stented. The first seven QRS complexes (\wedge) are wide (0.14 sec) with an abnormal morphology that does not resemble either a typical right or left bundle branch block. The RR intervals are regular at a rate of 74 bpm, and there are no P waves before any of the QRS complexes. The axis is indeterminate, between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). There is a P wave seen before the seventh complex (*) with a short PR interval (0.08 sec), best seen in the lead V1 rhythm strip. Noted in the lead II rhythm strip is that the initial portion of the QRS complex is different than in the preceding complexes. This is the superimposed P wave. There is also a P wave before the eighth QRS complex (+) with a PR interval of 0.16 second. The ninth to 13th QRS complexes are narrow (0.08 sec) and are preceded by a P wave (\blacktriangle) with a constant PR interval (0.22 sec). The P wave is upright in leads II and V4-V6; hence these are sinus complexes at a rate of 88 bpm.

Of note is that the morphology of the seventh and eighth QRS complexes (\uparrow) is different than the morphology of both the initial wide QRS complex and the narrow QRS complexes that follow, and the PR interval is shorter than seen with the sinus complexes. The seventh and eighth complexes are thus fusion complexes whose morphology results from antegrade ventricular activation through the AV node fusing with the complex generated by direct ventricular activation. The presence of fusion complexes confirms that the wide complexes are ventricular in origin (*ie*, an impulse generated from above fusing with an impulse generated from below). The ventricular rhythm is termed an accelerated idioventricular rhythm (AIVR) or slow ventricular tachycardia.

An AIVR results from an ectopic ventricular focus that generates an impulse faster than that generated by the sinus node (which may be due to an acceleration of the ventricular focus or slowing of sinus node activity). In this situation, when the sinus rate increases to a level faster than the rate of the AIVR, the AIVR is overdriven or suppressed and sinus rhythm is restored.

An AIVR is often the result of coronary reperfusion (spontaneous, catheter based, or thrombolytic) after a myocardial infarction (*ie*, a reperfusion arrhythmia). It has indeed been considered a marker of successful reperfusion. It is usually transient and generally does not require any therapy when there are no symptoms, as is often the case. If there are symptoms, the arrhythmia can be suppressed with an antiarrhythmic agent. However, it is important to distinguish between an AIVR and complete heart block with an escape ventricular focus. In complete heart block with an escape ventricular focus the atrial rate is faster than the ventricular rate, while with an AIVR the atrial rate is slower than the ventricular rate. The difference may also be established by seeing the onset of the ventricular arrhythmia. If the ventricular rhythm begins with an early ventricular QRS complex then the rhythm is an AIVR, while if the ventricular rhythm begins after a nonconducted P wave and pause, complete heart block is the etiology. Knowing the etiology is important because if there is complete heart block, an antiarrhythmic agent may suppress the escape ventricular focus, resulting in asystole. In this situation an antiarrhythmic agent is not administered. However, if arrhythmic suppression is necessary, a temporary pacemaker electrode should be inserted.

continues



ECG 51C Analysis: Normal sinus rhythm, T-wave inversions in leads V1-V4

ECG 51C, obtained 1 day after catheterization, shows that the ST-segment changes have resolved and the T waves are inverted in leads V1-V4 (↓), but they are asymmetric (slower in upstroke and faster in downstroke) and normal. There is a P wave (+) before each QRS complex with the same P-wave morphology, PR interval, and QRS morphology as seen in ECG 51A. The P waves are sinus in origin, and the sinus rate is 56 bpm. The QT/QTc intervals are normal (440/430 msec). Hence this is a normal sinus rhythm with T-wave inversion but no Q waves of the anterior wall, suggesting that reperfusion was successful and it is likely that there was no significant transmural myocardial damage. ■

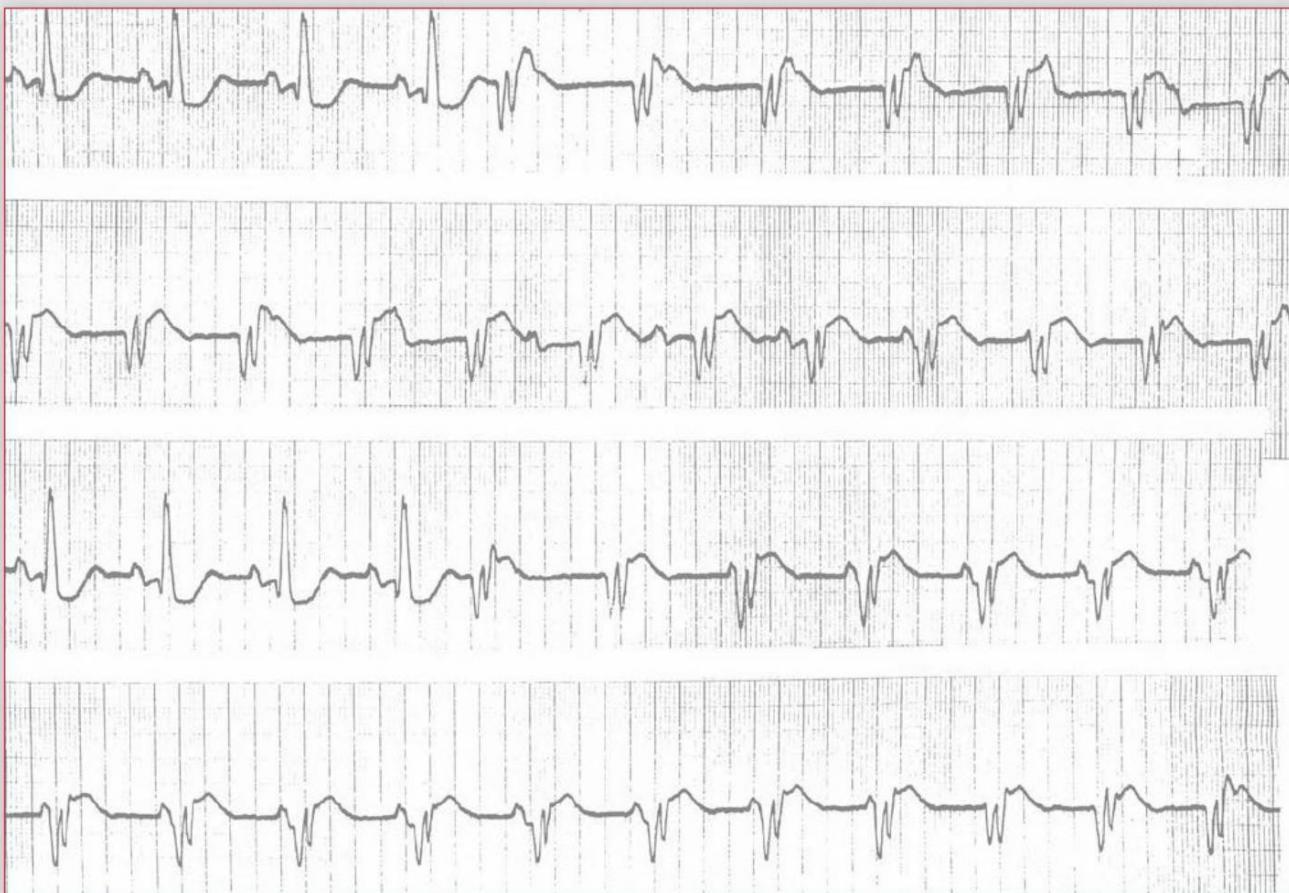
Notes

A 77-year-old woman with a history of heart failure and chronic kidney disease presents to the emergency department with intermittent lightheadedness and a throbbing sensation in her neck. She has dilated cardiomyopathy with a left ventricular ejection fraction of 35% and was recently started on digitalis for significant heart failure symptoms (New York Heart Association class III). The patient is placed on telemetry and a tracing is recorded while symptoms are present.

What is the rhythm disturbance?

What in particular is causing the throbbing in her neck?

What do you expect to find on physical examination?





ECG 52 Analysis: Sinus rhythm, AV dissociation with accelerated idioventricular rhythm

A series of rhythm strips recorded from telemetry are shown. The first two are continuous, as are the second two. Noted on the first strip are four regular QRS complexes with a slightly prolonged duration (0.12 sec); each QRS complex is preceded by a P wave (+) with a constant PR interval (0.20 sec). This is, therefore, a sinus rhythm at a rate of 60 bpm. However, the fifth QRS complex (↑), which occurs early, has a different morphology and is not preceded by a P wave. However, by measuring the PP intervals, it can be seen that there is an on-time P wave at the end the fifth QRS complex (*), giving the appearance of an R' waveform. The subsequent QRS complexes, which have the same morphology as the fifth complex, are regular at a rate of 70 bpm. Although P waves are not obvious, there are irregularities of the T waves and ST segments due to embedded or superimposed P waves (↓). Noted on the second strip are regular P waves (Λ) at a rate of 60 bpm, identical to the initial sinus rate. The P waves are dissociated from the QRS complexes as there is variability of the PR interval (↔), and occasionally the P wave is superimposed on or within the QRS complex. Hence there is AV dissociation. As the atrial rate is slower than the ventricular rate, the AV dissociation is due to an accelerated idioventricular ventricular rhythm.

The bottom two strips show the same pattern of four QRS complexes preceded by a P wave (+) with a constant PR interval followed by regular QRS complexes that differ from the sinus QRS complex and P waves (↓) that are dissociated from the QRS complex; that is, there is minor variability of the PR interval. However, in these strips the atrial rate is equivalent to the ventricular rate (*ie*, 62 bpm). Hence this is an idioventricular rhythm with isorhythmic dissociation; that is, there is AV dissociation present but the atrial and ventricular rates are

identical. In this situation the etiology for the AV dissociation cannot be reliably established. With complete or third-degree AV block the atrial rate is faster than the ventricular rate, while with an accelerated lower pacemaker (in this case ventricular) the atrial rate is slower than the rate of the QRS complexes. When the rates of the P waves and QRS complexes are the same, the etiology is not clear and hence the term isorhythmic dissociation is used.

Given chronic renal disease, this rhythm is very possibly the result of toxic effects of the recently initiated digitalis. Digitalis toxicity is associated with depression of the normal pacemaker tissue (sinus and AV nodal) by enhanced vagal tone as well as augmentation of outputs from the central sympathetic nervous system, which occur with digoxin toxicity. Hence as sinus and AV nodal depression occurs, the acceleration of a myocardial focus by sympathetic stimulation can result in an accelerated ventricular rhythm.

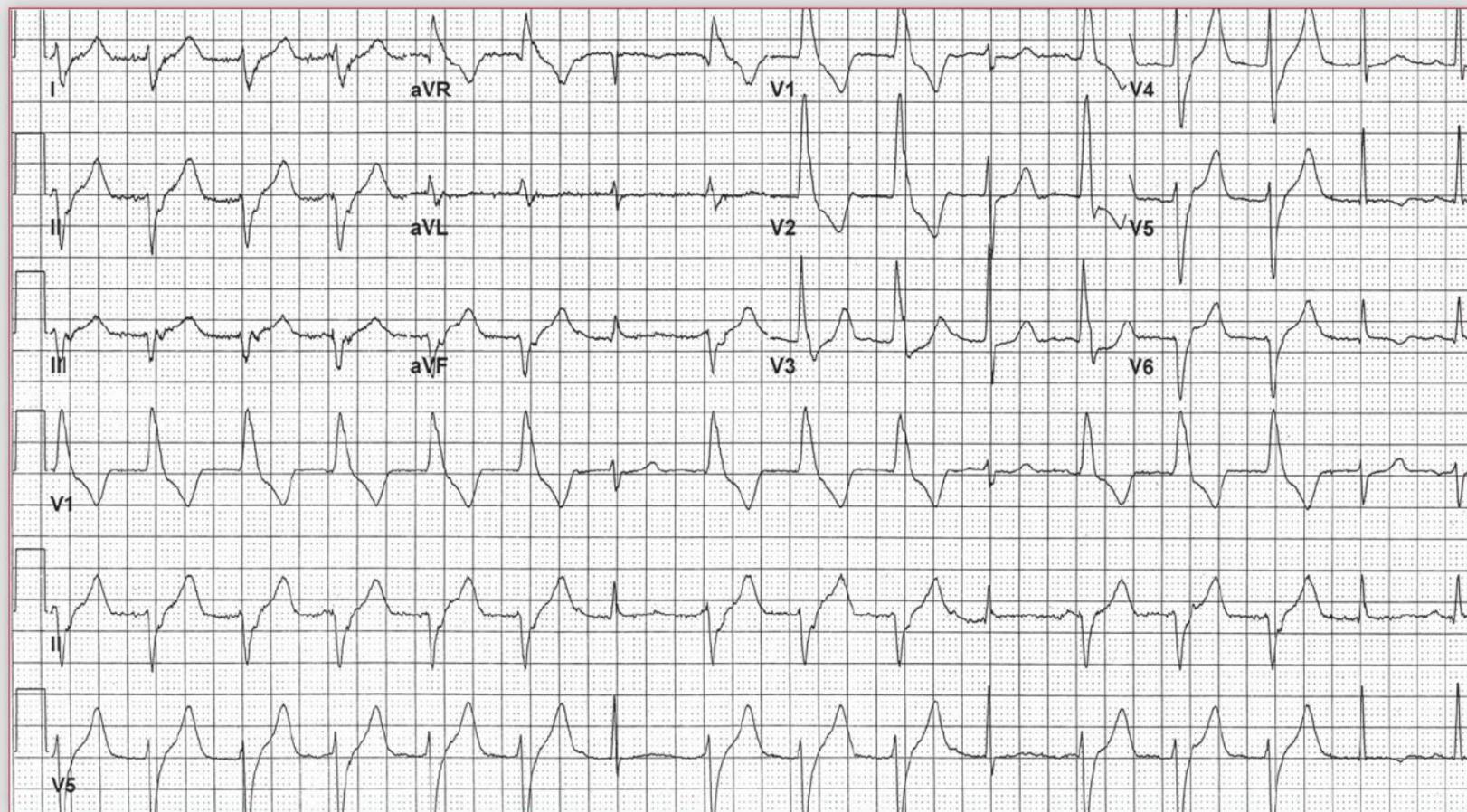
Common physical exam findings associated with AV dissociation and an accelerated ventricular rhythm include:

- Variable intensity of the peripheral pulse due to beat-to-beat variability in stroke volume resulting from changes in the relationship between atrial and ventricular contraction
- Cannon a waves, resulting from occasional atrial contraction against a closed tricuspid valve (The cannon a waves can be associated with throbbing sensations in the neck.)
- Variable intensity of S1 due to varying degrees of mitral and tricuspid valve closure at the time of ventricular systole ■

Core Case 53

You are consulted about a patient who has had intermittent episodes of exertional chest discomfort and dyspnea over the past 24 hours. She has diabetes, hypertension, and a family history

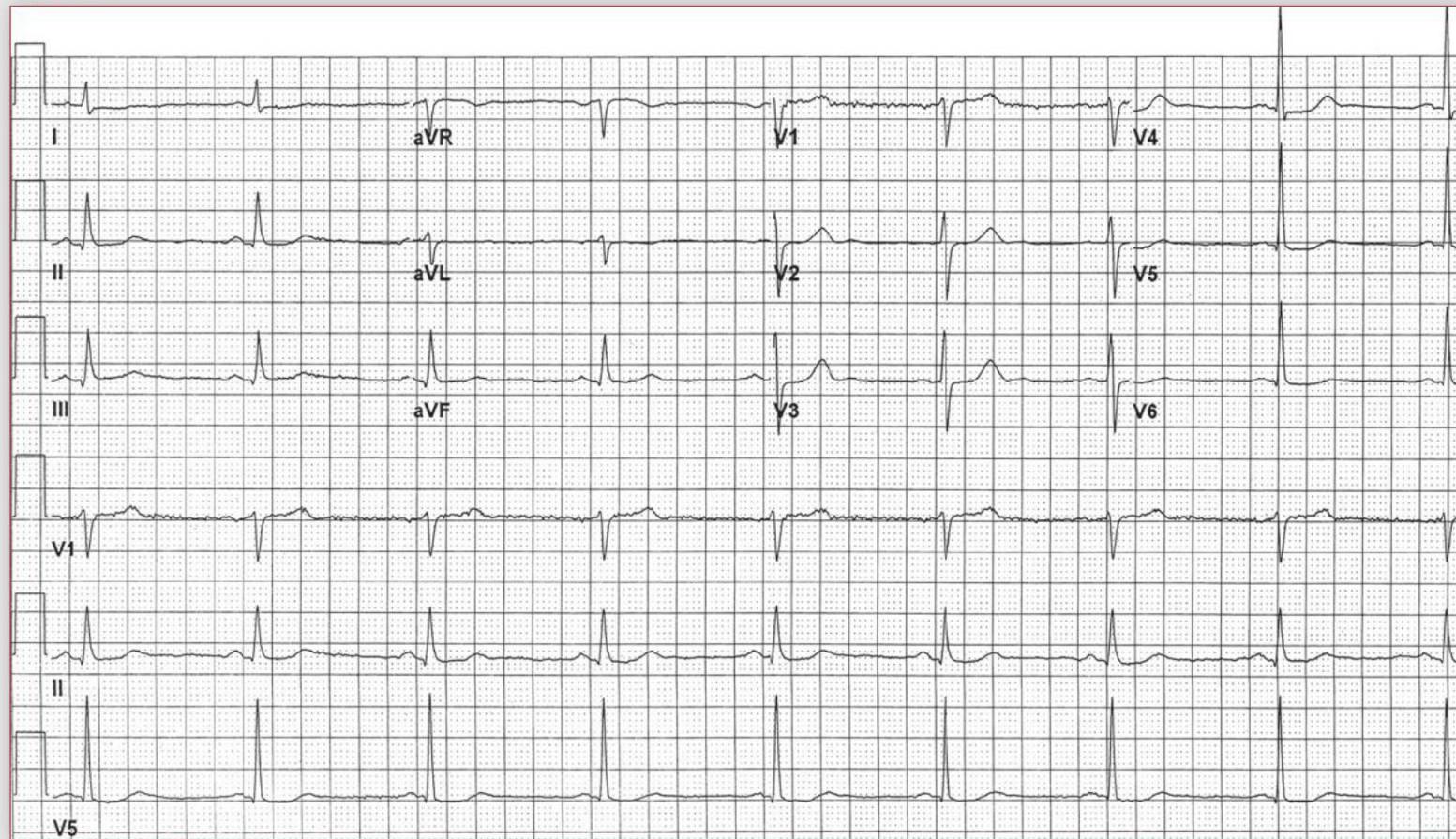
ECG 53A

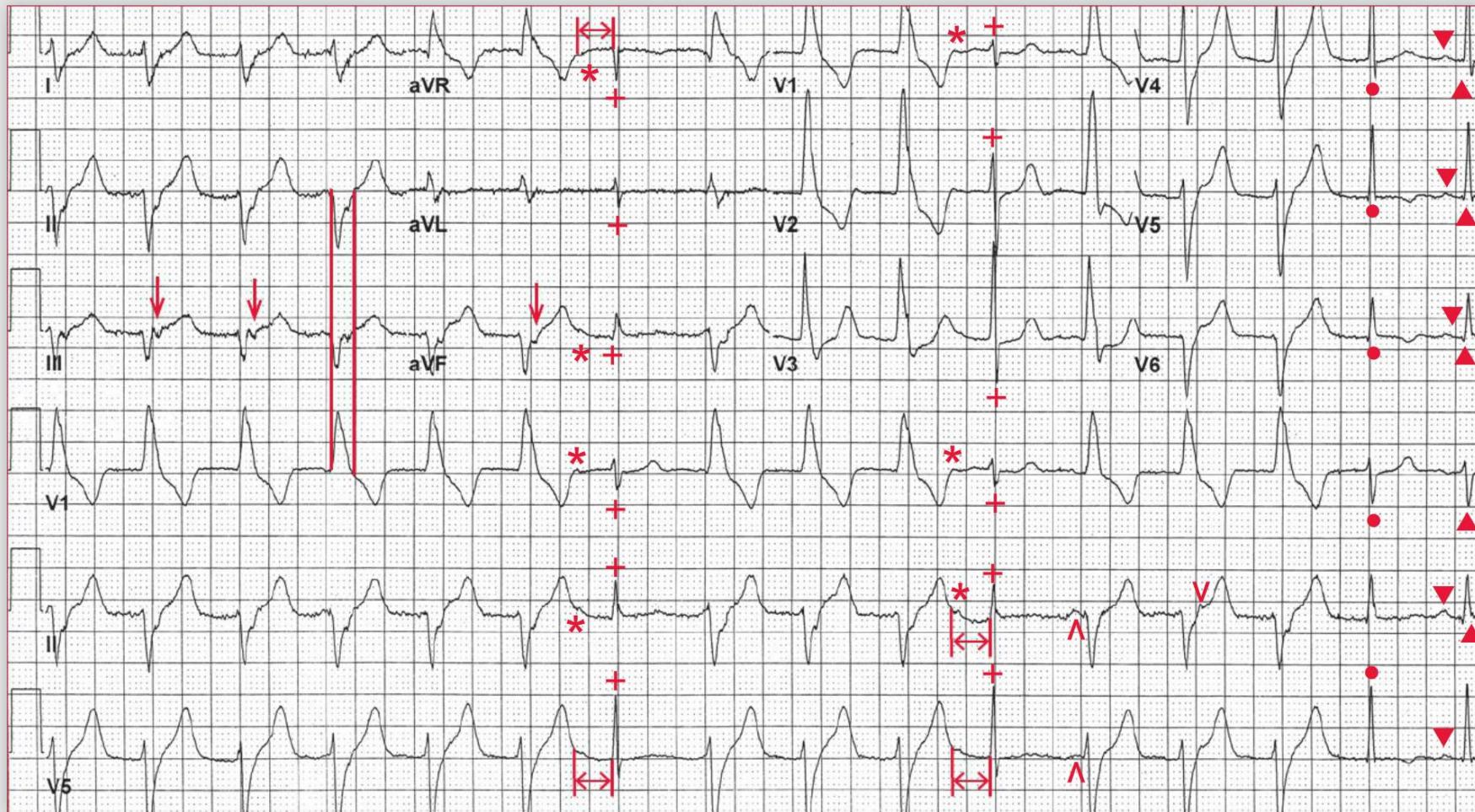


of coronary artery disease. Physical examination demonstrates irregularities in both heart sounds and pulse. An ECG is obtained (53A) and compared with an ECG obtained a year earlier (ECG 53B).

What is the origin of the wide QRS complexes?
How would you manage this patient's rhythm abnormality?

ECG 53B





ECG 53A Analysis: AV dissociation with accelerated idioventricular rhythm and frequent capture complexes (Dressler complexes)

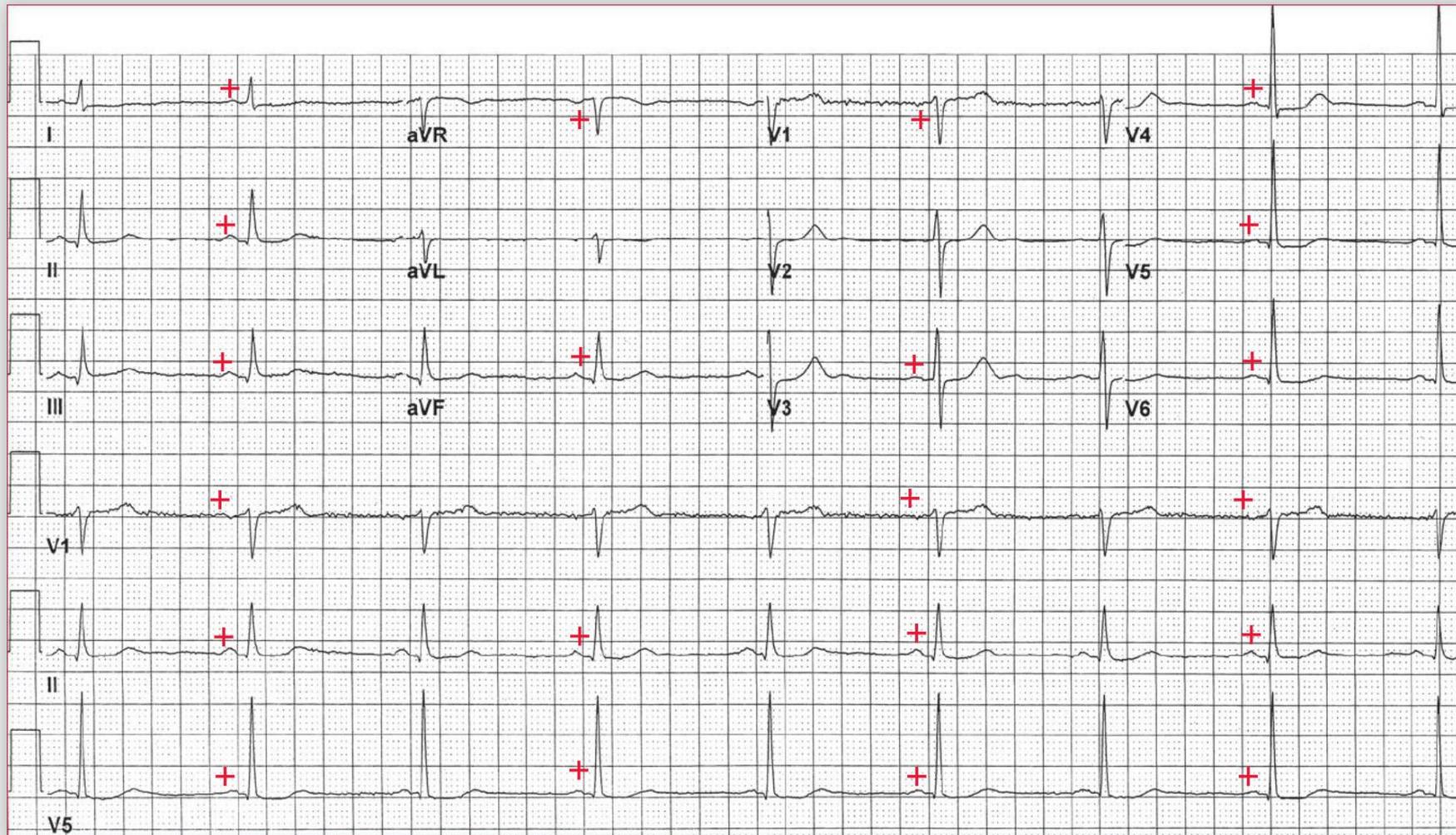
ECG 53A shows a regular rhythm at a rate of 92 bpm. There are two different QRS complex morphologies present. Most of the QRS complexes are wide with a duration of 0.16 second. These complexes are not preceded by a P wave, and they have an abnormal morphology (neither typical right nor left bundle branch block) and an indeterminate axis, between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). These features are consistent with a ventricular origin. Importantly, an indeterminate axis associated with a wide QRS complex is a result of direct myocardial activation as is seen with a paced complex (biventricular pacing), a complex due to Wolff-Parkinson-White, or a ventricular complex. Although notches can be seen at the end of the QRS complex in leads III and aVF (\downarrow), these are part of the QRS complexes, which is confirmed by measuring the maximal QRS width and comparing it with the QRS complex width as measured in these leads (\parallel).

There are four narrow QRS complexes (+, ●, ▲) with a duration of 0.08 second (complexes 7, 11, 15, and 16). There are P waves (*) before complexes 7 and 11, and the PR interval (\leftrightarrow) is the same for each complex (0.26 sec). These are, therefore, conducted sinus complexes. Careful inspection of the lead II rhythm strip demonstrates the presence of atrial activity (A) prior to the 12th QRS complex, which is wide and has the same morphology as all of the other wide QRS complexes. The PR interval is shorter (0.12 sec) than the conducted P wave of complexes 7 and 11. Hence this P wave is not likely conducted. In addition, there is notching in the ST segment (v) after the 13th complex. This is due to a superimposed P wave and indicates that there is underlying atrial activity that is dissociated from the QRS complex.

Based on the presence of several sequential P waves, it can be seen that the atrial rate is 75 bpm. The presence of AV dissociation with an atrial rate slower than the ventricular rate indicates an accelerated lower pacemaker focus. The lower focus is ventricular in origin (and not junctional) since the captured complexes (+) are narrow with a normal morphology. The narrow complexes are captured and are termed Dressler complexes. The presence of either fused beats or completely captured beats (Dressler complexes) confirms AV dissociation. AV dissociation with a wide complex rhythm is the hallmark for a ventricular rhythm. Hence this is an accelerated idioventricular rhythm (AIVR) or slow ventricular tachycardia.

The last two QRS complexes are also narrow. While there is no obvious atrial activity prior to the 15th (next to last) QRS complex (●), it is likely hidden within the preceding T wave. The last (16th) QRS complex (▲) is preceded by a P wave (▼) and has a PR interval of 0.16 second. This is a sinus complex. The difference in the PR intervals between the Dressler complexes (+) (0.26 sec), which are longer, and this sinus complex (▲) (0.16 sec) is a result of retrograde concealed conduction into the AV node. The ventricular complexes result in retrograde activation of the AV node, which accounts for the AV dissociation as the AV node is generally refractory and does not conduct the sinus impulse in an antegrade fashion. However, if an appropriately timed sinus impulse (occurring later after the ventricular complex) enters the AV node antegradely at a time when there has been partial recovery of AV nodal conduction (*ie*, the AV node is not completely refractory), it may conduct the sinus impulse, although at a slower rate

continues



ECG 53B Analysis: Normal sinus rhythm

since it is partially refractory. This results in a captured QRS complex (Dressler complex) with a longer PR interval than that of the sinus complex. This is a form of retrograde concealed conduction resulting from the ventricular complex.

Intermittent AV conduction with fusion complexes and Dressler complexes is seen with AV dissociation and is most often present when the ventricular rate is slower, allowing more time for antegrade conduction through the AV node.

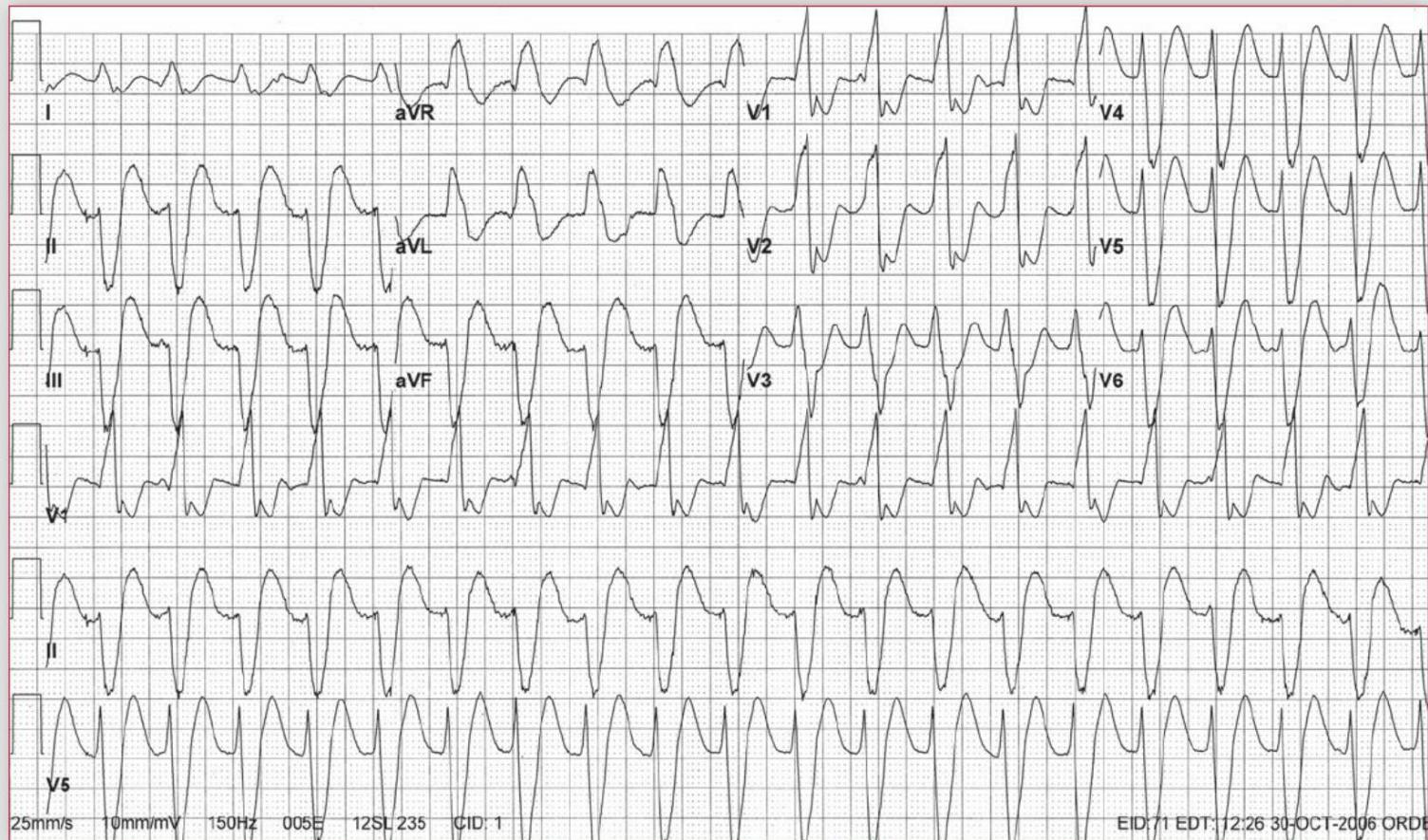
Given her history of intermittent chest pain, this patient should undergo a thorough evaluation for ischemia since AIVR is often associated with reperfusion of a coronary artery (either spontaneous or related to treatment with angioplasty or thrombolysis). It is, therefore, possible that there is intermittent occlusion of a vessel with spontaneous reperfusion. If no evidence of obstructive coronary disease is found, then attempts at controlling the AIVR (particularly if it is associated with symptoms) can be made with either a β -blocker or anti-arrhythmic therapy.

In ECG 53B the rhythm is regular at a rate of 50 bpm. The QRS complex duration (0.08 sec) and morphology are normal and the axis is normal, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (460/420 msec). There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The QRS complex duration and morphology are identical to those of the narrow captured QRS complexes (Dressler complexes) seen in ECG 53A. The PR interval is the same as the last sinus complex (\blacktriangle) in ECG 53A. This confirms that these narrow complexes seen in ECG 53A are the result of a sinus impulse that conducts through the AV node. ■

Core Case 54

A 65-year-old man with known coronary disease presents with lightheadedness and palpitations. His initial ECG is ECG 54A. ECG 54B is his baseline ECG.

ECG 54A



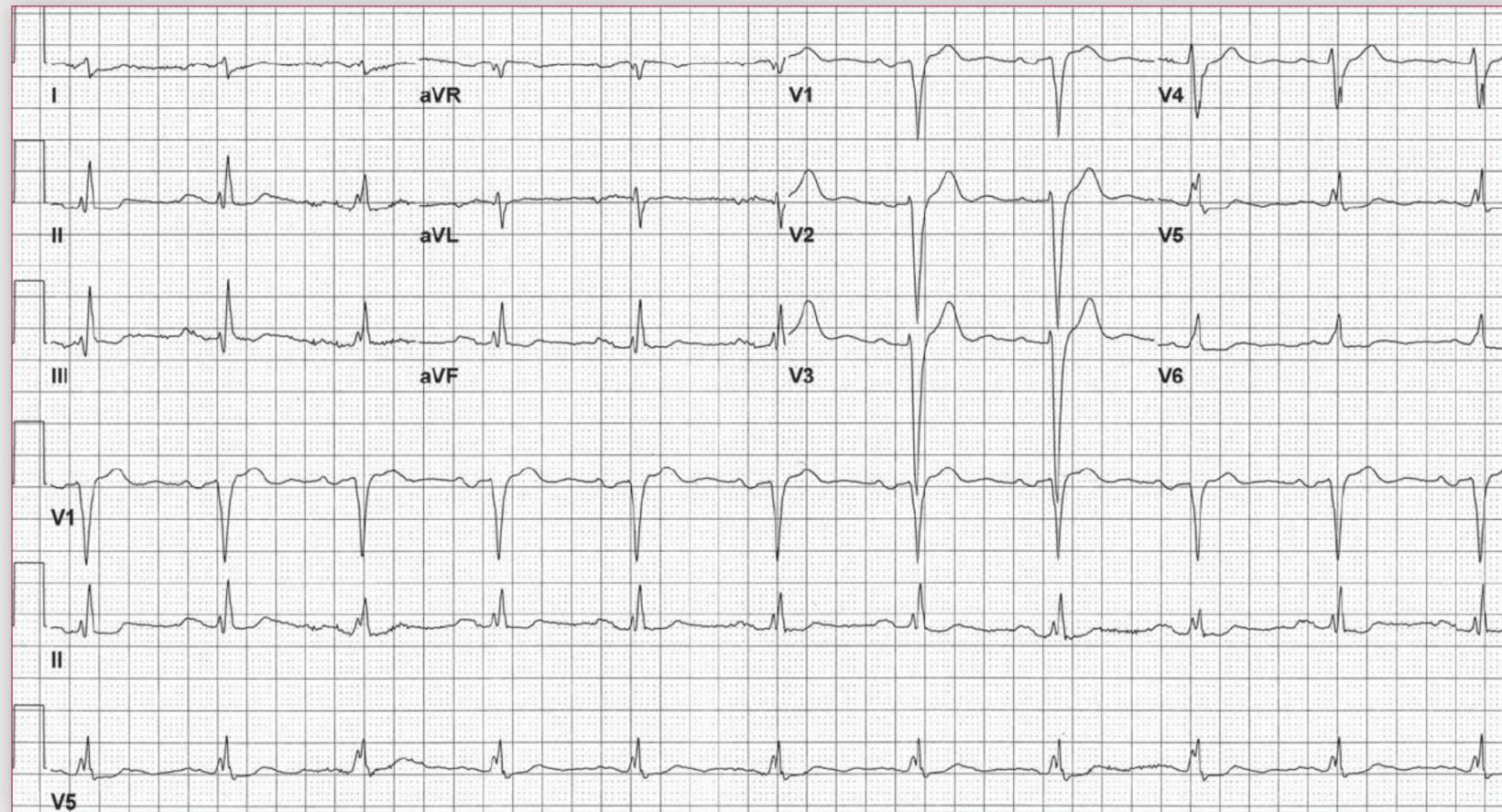
What is the rhythm abnormality?

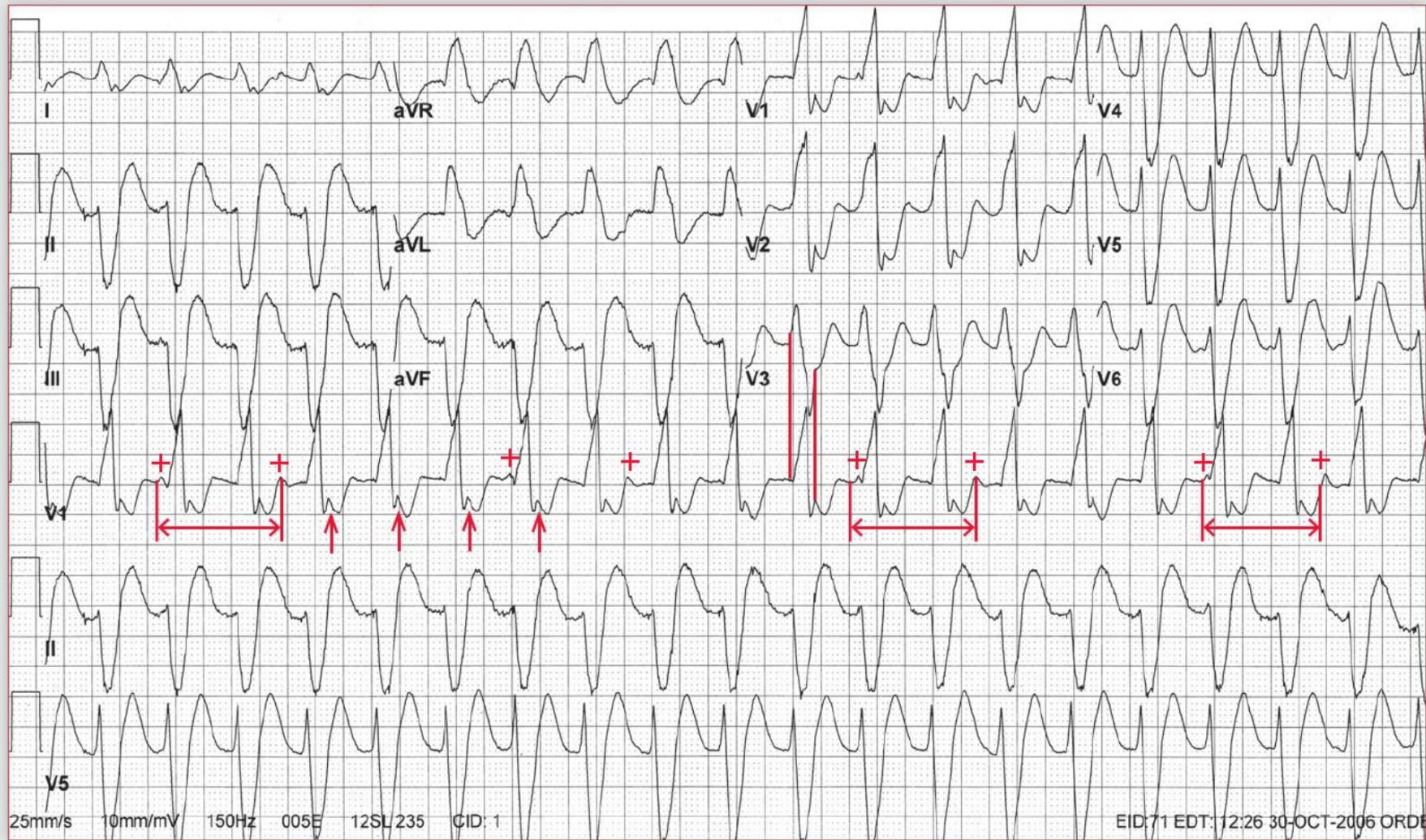
What is the etiology for the arrhythmia?

What ECG feature(s) favor this diagnosis?

What ECG feature(s) make this diagnosis definitive?

ECG 54B





ECG 54A Analysis: Sustained monomorphic ventricular tachycardia

In ECG 54A the rhythm is regular at a rate of 130 bpm. The QRS complex is wide (0.20 sec). The QRS morphology is abnormal and does not resemble either a typical right or left bundle branch block. Although no obvious P waves are seen before any of the QRS complexes, there is evidence of atrial activity (+), best seen in the lead V1 rhythm strip, for example, before the second, seventh, 12th, and 17th QRS complexes and after the third, eighth, 13th, and 18th QRS complexes (note the positive waveform at the end of the T wave that is not seen with the other QRS complexes). It should be noted that the positive waveform that appears to be present after the QRS complex in leads V1-V2 (↑) is not a P wave but is actually part of the QRS complex. This can be established by measuring the maximum QRS complex width (*eg*, in lead V3) and comparing this with the QRS complex width in leads V1-V2 (||).

There is no relationship between the P waves and the QRS complexes (*ie*, the PR intervals are variable without any pattern); therefore, this is AV dissociation. It is not necessary that the P waves “march out” completely to make the diagnosis of AV dissociation, but rather that there are P waves associated with some (but not all) of the QRS complexes. The atrial rate is about 72 bpm, which can be established by the fact that whenever two sequential P waves are seen (↔) the rate

is identical at 72 bpm. A wide complex tachycardia associated with AV dissociation and an atrial rate that is slower than the ventricular rate (*ie*, an enhanced lower focus) is characteristic of sustained ventricular tachycardia. As all the QRS complexes are similar, this is termed monomorphic ventricular tachycardia. AV dissociation occurs because the ventricular complex conducts into the AV node but does not get through to activate the atrium. However, it does totally depolarize the node, causing it to be completely refractory and preventing antegrade conduction of the sinus complex. Since the ventricular impulse does not reach the atrial myocardium, the sinus node is not depressed or overdriven.

More than 80% of wide complex tachycardias are ventricular in origin, and the percentage is even higher (> 90%) in patients with structural heart disease such as prior myocardial infarction. Sustained monomorphic ventricular tachycardia is not provoked by active ischemia but is commonly seen in ischemic heart disease with prior myocardial damage and scar. The mechanism is most often reentry, due to a reentrant circuit that results from a myocardial scar (scar-dependent) surrounded

continues

by normal myocardial tissue. Hence the reentrant circuit involves viable Purkinje fibers or pathways located within scar tissue as well as Purkinje fibers or pathways that are within the viable myocardium around the scar. These Purkinje pathways have different conduction properties and different refractory periods. They are linked within the ventricular myocardium, forming a circuit.

It is often challenging to distinguish ventricular tachycardia from supraventricular tachycardia with aberrancy. Supraventricular tachycardia may originate from the sinus node, atrial myocardium, or AV node or junction. The wide complex may be due to either a rate-related or functional bundle branch block or to underlying conduction system disease in which the bundle branch block is present at baseline during normal sinus rhythm. Other causes for wide complex tachycardia are rhythms that are associated with a pacemaker (the pacemaker tracking an atrial arrhythmia or pacemaker-mediated tachycardia), a sinus or atrial arrhythmia associated with Wolff-Parkinson-White, or an antidromic atrioventricular reentrant tachycardia due to Wolff-Parkinson-White.

The initial evaluation of a patient with wide complex tachycardia includes an assessment of vital signs and level of consciousness. Regardless of the etiology, a patient with wide complex tachycardia who is hemodynamically unstable should receive immediate electrical cardioversion and subsequent treatment based on the Advanced Cardiovascular Life Support algorithms. The hemodynamic consequences of

the tachycardia are not related to its etiology but rather to the rate of the tachycardia as well as the nature and extent of underlying heart disease.

There are a number of ECG features that are useful in establishing the etiology as either ventricular tachycardia or supraventricular tachycardia with aberration:

- The presence of AV dissociation (*ie*, variable PR intervals with no relationship between P waves or QRS complexes) and a ventricular rate faster than the atrial rate are the most important features of ventricular tachycardia. It is rare for supraventricular tachycardia to have AV dissociation. Supporting a diagnosis of AV dissociation is the presence of fusion or captured QRS complexes (also called Dressler complexes). Fusion or captured QRS complexes are the result of antegrade conduction of an impulse through the AV node that fuses with an impulse generated by the ventricular complex and that results in partial (fusion) or complete ventricular capture. This occurs more often when the rate of the ventricular tachycardia is slower, causing less retrograde conduction into the AV node from the ventricular complex and allowing for more antegrade conduction through the node of the sinus impulse.

- In supraventricular tachycardia, regardless of the etiology (*ie*, sinus, atrial, or AV nodal), activation of the ventricle is always via the same pathway, which may be the normal AV node–His-Purkinje system or an accessory pathway. As the activation sequence is always the same, all the QRS complexes are identical to each other. In addition, all the ST-T waves are identical. In contrast, ventricular tachycardia is due to a small circuit within the ventricular myocardium and ventricular activation bypasses the normal Purkinje system. As the vector or pattern of ventricular activation may change, there may be changes in the direction of ventricular activation or the myocardial activation sequence. Hence there may be subtle differences in the morphology of the QRS complexes or ST-T waves. Differences in ST-T-wave morphology may also be the result of AV dissociation with superimposed P waves.
- An indeterminate axis (negative QRS complex in leads I and aVF) associated with a wide QRS complex occurs only when there is direct ventricular activation, bypassing the normal His-Purkinje system. Conduction through the normal His-Purkinje system, even if there is aberration, will not be associated with an indeterminate axis. Hence this is seen with a ventricular, paced (particularly biventricular pacing), or preexcited (as with Wolff-Parkinson-White pattern) complex.
- Positive QRS complex concordance in the precordial leads (*ie*, tall R waves in leads V1-V6) is only seen when there is direct ventricular myocardial activation (*ie*, ventricular, paced, or preexcited QRS complex) in which the normal His-Purkinje system is bypassed. No form of aberration associated with a supraventricular rhythm in which conduction is through the normal His-Purkinje system will produce positive QRS complex concordance. In contrast, negative QRS complex concordance (*ie*, deep QS complexes in leads V1-V6) can be seen with a typical left bundle branch block pattern. Hence negative concordance is not useful.
- A QRS complex wider than 160 msec is uncommonly seen with a bundle branch block and is most often associated with a ventricular complex. Exceptions are dilated cardiomyopathy (with diffuse fibrosis and a pronounced intraventricular conduction delay) and the presence of hyperkalemia, which may cause widening of a supraventricular QRS complex (due to a marked slowing of impulse conduction) and may be associated with a QRS complex width that exceeds 160 msec. A QRS complex wider than 240 msec is primarily due to hyperkalemia.

continues

- A significant shift in the QRS complex axis, particularly to the left, suggests, but is not diagnostic for, ventricular tachycardia. A shift of the axis rightward or a normal axis does not favor one diagnosis over another.
- In general, aberration is due to a terminal delay in ventricular activation (*ie*, either a right or left bundle branch block results in delayed activation of the ventricle innervated by the inactive or blocked bundle). The initial forces of the QRS complex are, however, normal in width and morphology as the initial ventricular activation still occurs via the normally conducting bundle and Purkinje system. In contrast, ventricular tachycardia is due to activation that does not use the normal Purkinje system, but rather there is direct myocardial stimulation. Direct myocardial stimulation results in slow conduction and hence the entire QRS complex, including the initial portion, is wide, reflecting diffuse slowing of impulse conduction. Hence, if in any precordial lead there is an RS complex, an R/S ratio less than 1 (*ie*, the complex is wide as a result of a widened terminal portion or S wave), or an R-wave width less than 100 msec, then the initial ventricular activation time is normal and this strongly suggests aberration as the cause of the wide QRS complex. In

contrast, if the R/S ratio exceeds 1 or the R-wave width is greater than 100 msec, there is abnormal initial ventricular activation and this strongly suggests a ventricular complex. This criterion, however, may not be valid in the setting of severe dilated cardiomyopathy, in which diffuse fibrosis is causing all of ventricular activation to be slow, or with Wolff-Parkinson-White, in which there is also direct initial myocardial activation via the accessory pathway.

- Specific QRS morphology criteria in leads V1 and V6 are less useful as they may not be definitive, although they may suggest a specific etiology. Such relationships are generally based on a statistical correlation, and hence there is a good deal of overlap. Importantly, morphologic criteria that might favor a ventricular complex may be seen when there is a significant intraventricular conduction delay (IVCD) present during sinus rhythm, limiting their usefulness. Moreover, they are less valid when trying to distinguish between a ventricular and a preexcited complex.

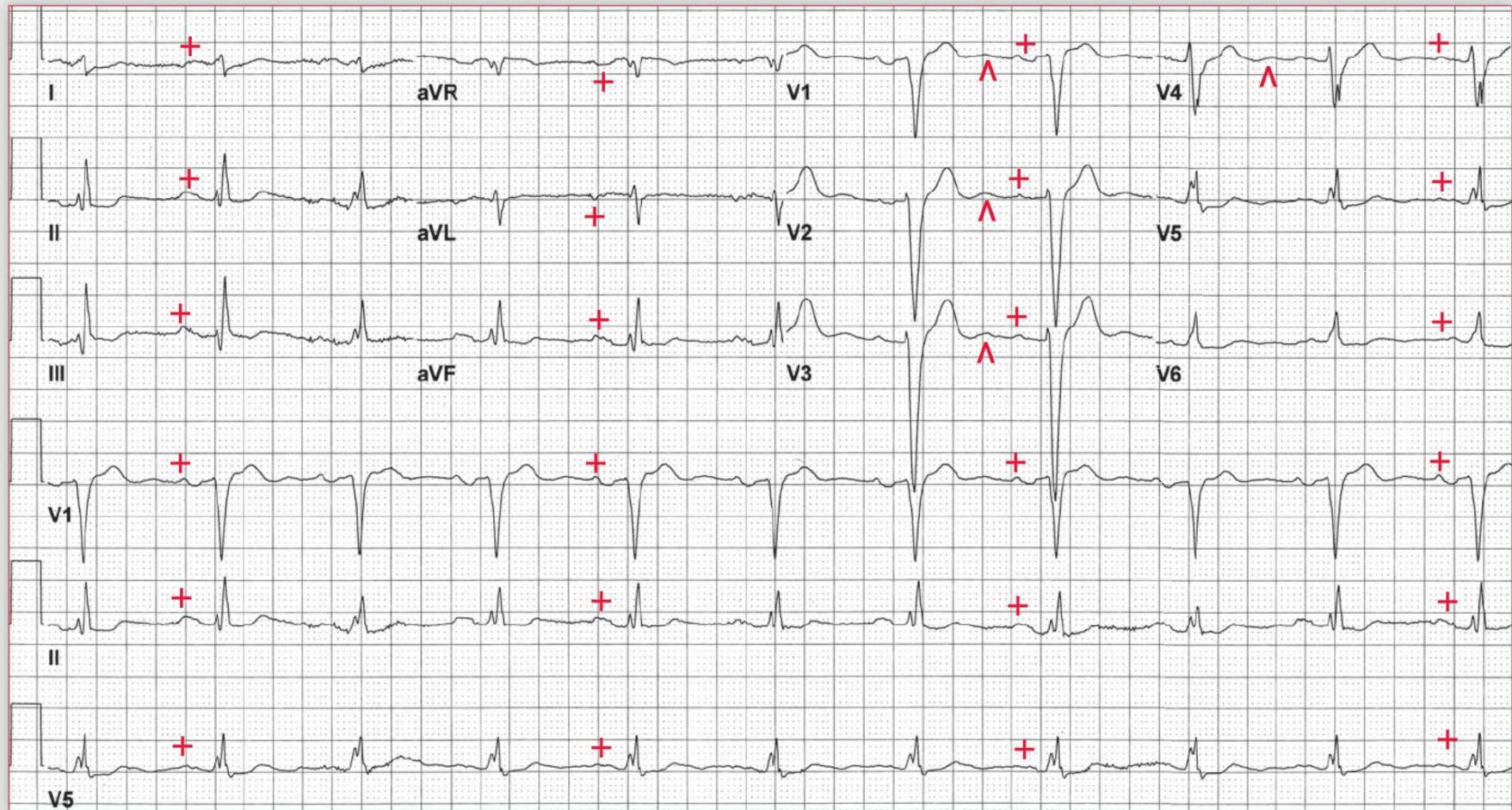
However, morphologic criteria that have been proposed include:
–A monophasic R or biphasic qR complex in lead V1 favors ventricular tachycardia; this represents the lack of an RSR' pattern.

- A triphasic RSR' or RSR' complex (the so-called “rabbit-ear” sign) in lead V1 usually favors supraventricular tachycardia. As an exception, if the R wave (initial positive waveform) of the RsR' complex is taller than the R' waveform (terminal positive deflection), then ventricular tachycardia is suggested.
- An rS complex (R wave smaller than S wave) in lead V6 favors ventricular tachycardia. In contrast, an Rs complex (R wave larger than S wave) in lead V6 favors supraventricular tachycardia.
- A broad initial R wave lasting 40 msec or longer in lead V1 or V2 favors ventricular tachycardia. In contrast, the absence of an initial R wave or a small initial R wave less than 40 msec in lead V1 or V2 favors supraventricular tachycardia.
- A slurred or notched downstroke of the S wave in lead V1 or V2 combined with a duration from the onset of the QRS complex to the nadir of the QS or S wave of 60 msec or longer in lead V1 or V2 favors ventricular tachycardia. In contrast, a swift, smooth downstroke of the S wave in lead V1 or V2 with a duration of less than 60 msec favors supraventricular complex.

- The presence of any significant Q wave or a QS complex in lead V6 is suggestive of ventricular tachycardia. In contrast, the absence of a Q wave in lead V6 favors a supraventricular complex.

In ECG 54A, the presence of AV dissociation and variability of the ST-T waves establishes the diagnosis of ventricular tachycardia. However, there are also other features characteristic of ventricular tachycardia, including a QRS complex wider than 160 msec, left axis (positive QRS complex in lead I and negative QRS complex in leads II and aVF), a wide initial broad and monophasic R wave (> 40 msec) in lead V1, and an R-wave width greater than 100 msec in leads V2-V3 accompanied by an R/S ratio less than 1.

continues



ECG 54B Analysis: Normal sinus rhythm, first-degree AV block, intraventricular conduction delay, left atrial hypertrophy (or left atrial abnormality), nonspecific ST-T wave abnormalities

ECG 54B shows a regular rhythm at a rate of 62 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4-V6; hence this is a sinus rhythm with first-degree AV block. The P wave is broad (0.18 sec), which is characteristic of left atrial hypertrophy or a left atrial conduction abnormality. The QRS complex duration is increased (0.14 sec), but the pattern is not typical for either a right or left bundle branch block. Hence this is a nonspecific IVCD. The QT/QTc intervals are normal (420/430 msec and 360/370 msec when corrected for

the prolonged QRS complex duration). There are diffuse nonspecific ST-segment abnormalities (*eg*, T-wave flattening). Noted are U waves (Δ) in leads V1-V4 that are a normal variant.

The QRS complex during sinus rhythm should be compared with that during ventricular tachycardia as is present in ECG 54A. The difference in QRS morphology, the presence of AV dissociation, and the ST- and T-wave variability are very obvious. ■

Core Case 55

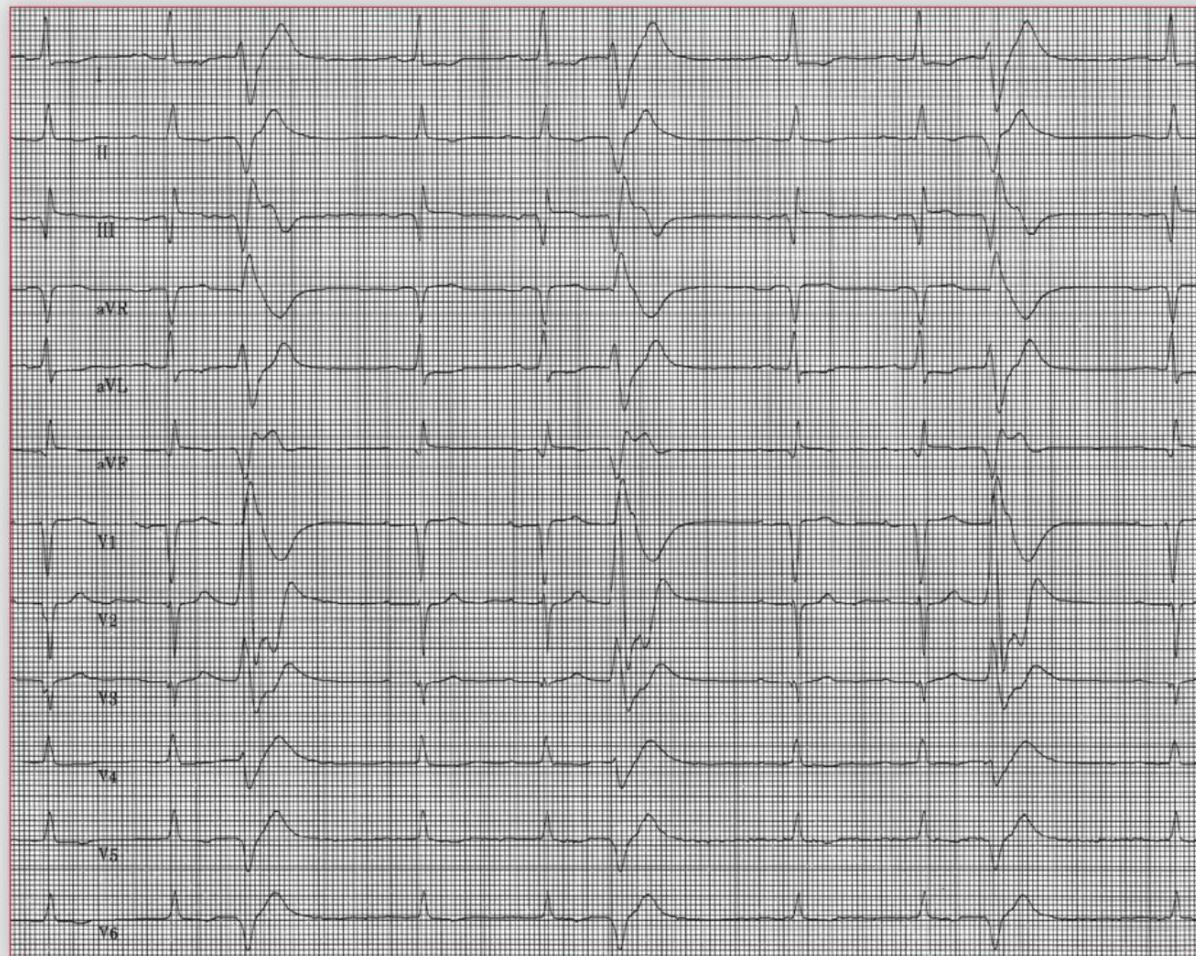
A 78-year-old woman with ischemic heart disease, prior myocardial infarction, and a left ventricular ejection fraction of 30% presents acutely to the emergency department with

ECG 55A



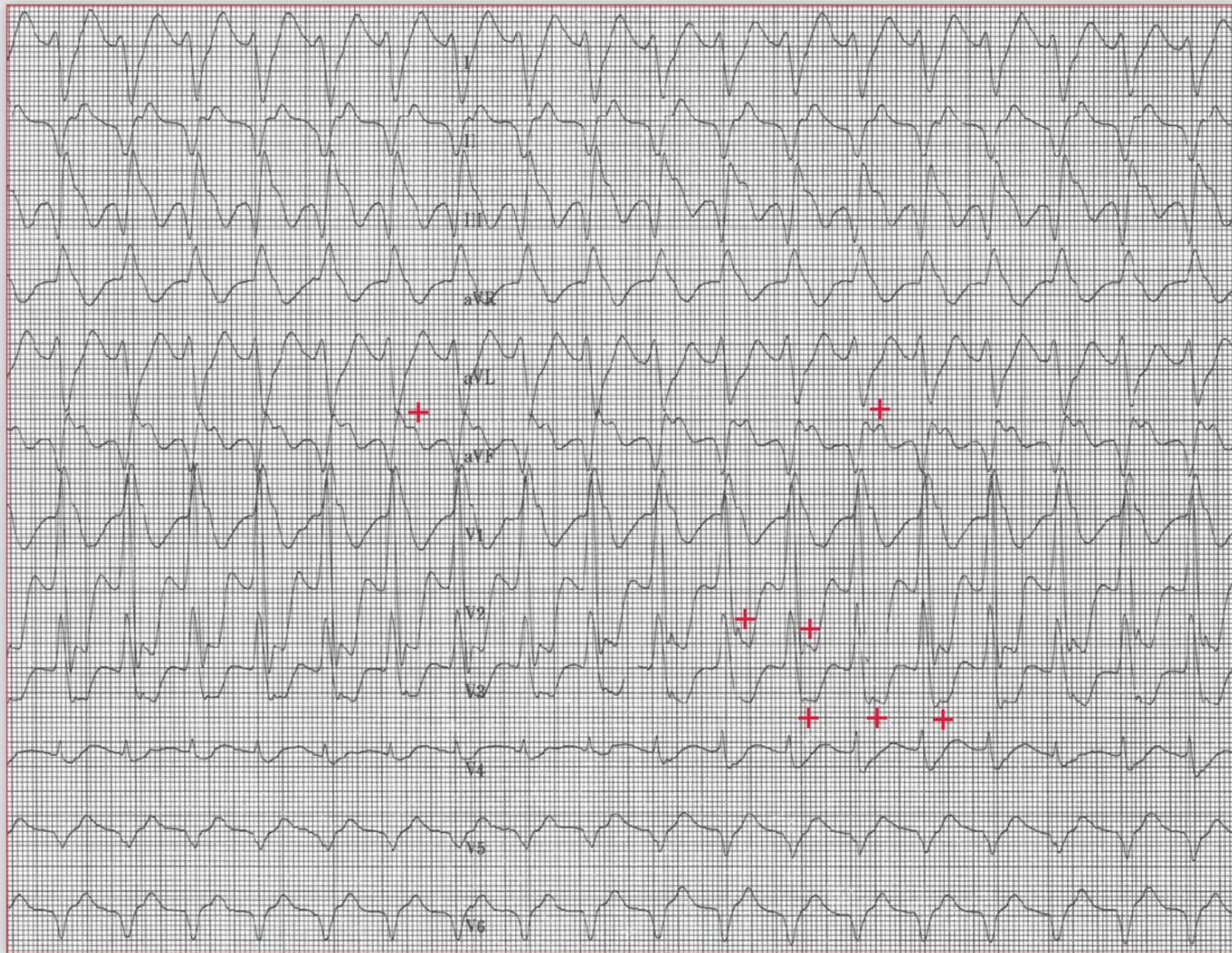
hypotension and altered mental status. Her blood pressure is 80/palp, and her pulse is regular. An ECG (55A) is obtained. Later during the woman's hospitalization a second ECG (55B) is obtained.

ECG 55B



What is the rhythm abnormality?

How would you manage this patient acutely?



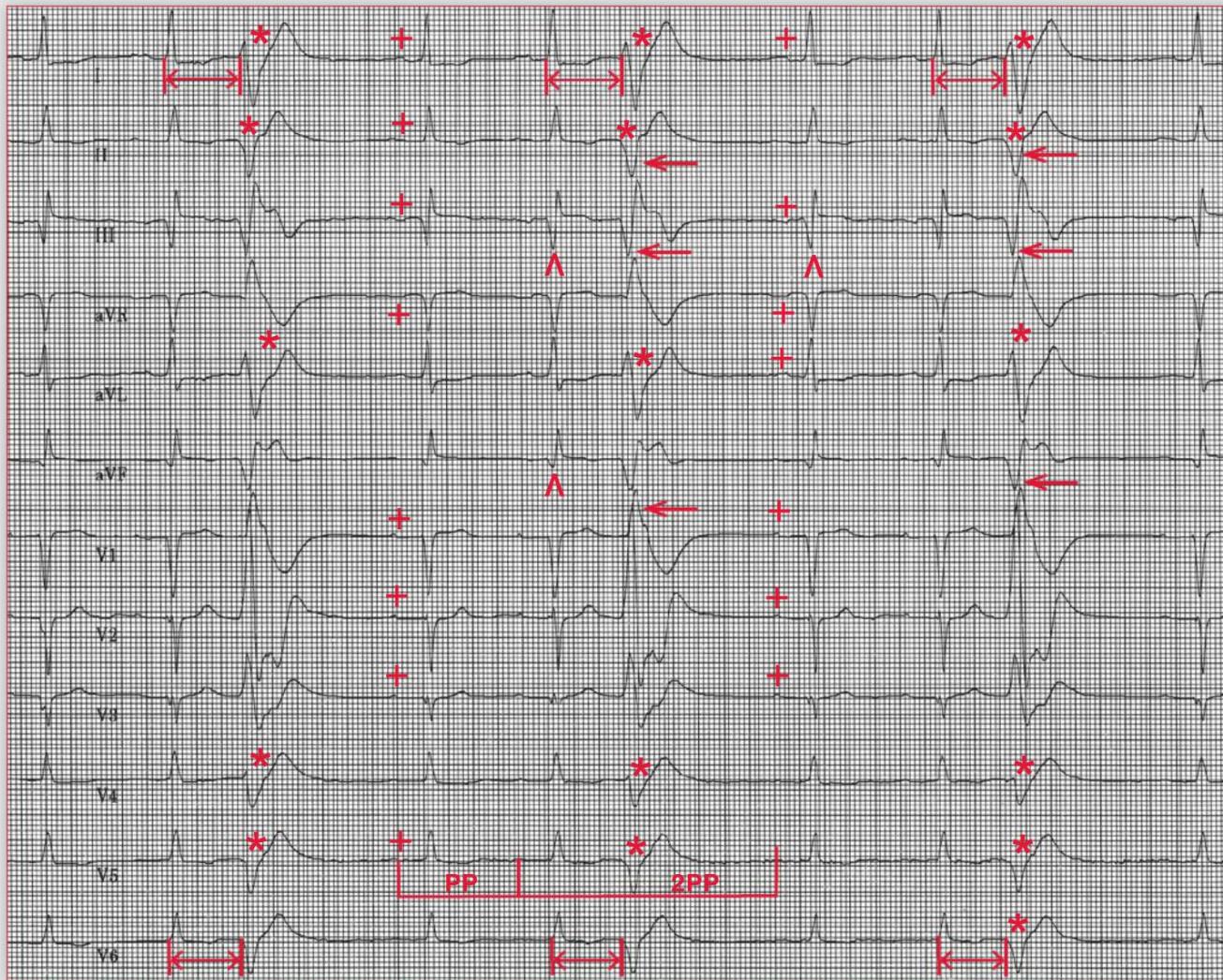
ECG 55A Analysis: Sustained monomorphic ventricular tachycardia

In ECG 55A, there is a regular wide complex tachycardia (QRS complex duration 0.18 sec) at a rate of 120 bpm. The axis is indeterminate, between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). There is no obvious atrial activity (*ie*, P waves) before any of the QRS complexes. The QRS complex morphology is abnormal, and although the QRS complex morphology resembles a right bundle branch block it is not a typical right or left bundle branch block. Along with the indeterminate axis, the lack of an RSR' pattern in lead V1 with a tall, broad R wave and the presence in lead V6 of an S wave that is deeper than the R wave is tall are consistent with the diagnosis of a ventricular rhythm. The segments (+) in leads aVF and V2-V3 show variability in morphology, occasionally suggesting a P wave. The absence of a P wave before any QRS complex, the abnormal QRS complex morphology and width, and the indeterminate axis are all consistent with the diagnosis

of sustained ventricular tachycardia. Because all of the QRS complexes are identical in morphology, this would be termed monomorphic ventricular tachycardia.

Given the unstable nature of the patient's presentation, immediate electrical cardioversion is indicated. This should be followed by identification of any reversible causes for her ventricular tachycardia such as electrolyte disturbances, drug toxicities, or enhanced sympathetic activity. Active ischemia is not a precipitating factor for sustained monomorphic ventricular tachycardia, although very commonly there is an abnormal substrate due to chronic ischemic heart disease (*ie*, the presence of a scar from previous myocardial damage). It is reasonable to initiate antiarrhythmic therapy with agents such as amiodarone or lidocaine to prevent recurrent episodes after cardioversion.

continues



ECG 55B Analysis: Normal sinus rhythm, first-degree AV block, ventricular trigeminy, old inferior wall myocardial infarction

The diagnosis of ventricular tachycardia in ECG 55A can be confirmed by comparison with ECG 55B, which shows a regular rhythm with occasional premature complexes (*). There is a fixed relationship (coupling interval) between the premature complexes and the sinus complexes (↔). Hence the rhythm is regularly irregular. The narrow QRS complexes have a normal interval (0.08 sec). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). Q waves are also seen in leads aVF and III (Δ), consistent with a prior inferior wall myocardial infarction. There is a P wave (+) before each QRS complex with a stable PR interval (0.26 sec). The P waves have a small amplitude, but they are positive in leads I, II, aVF, and V4-V5. Hence these are sinus complexes and there is first-degree AV block or prolonged AV conduction. The sinus rate is 60 bpm. The QT/QTc intervals are normal (400/400 msec).

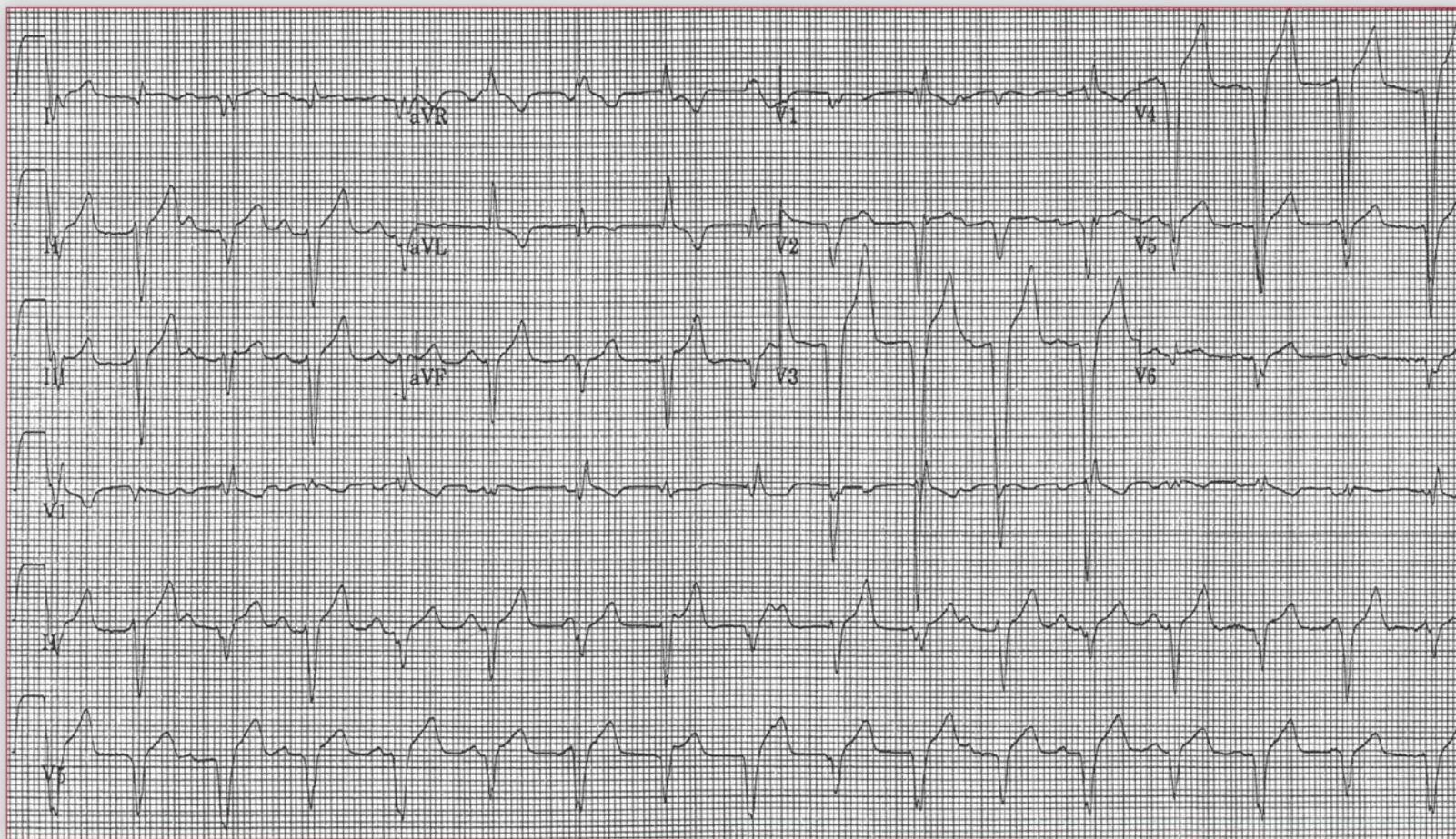
The premature complexes are wide (0.18 sec), are abnormal in morphology, and are not preceded by a P wave. They all have the same morphology, which does not resemble either a typical right or left

bundle branch block. These are unifocal premature ventricular complexes. They have a fixed relationship with the sinus complex that precedes them (*ie*, there is a fixed coupling interval). They are all associated with a full compensatory pause; that is, the PP interval surrounding the premature complex is equal to two sinus PP intervals (□). Every third QRS complex is a premature ventricular complex, which is termed ventricular trigeminy. Importantly, the premature ventricular complexes have exactly the same morphology in every lead as the QRS complexes of the tachycardia in ECG 55A, confirming the fact that the rhythm in ECG 55A is sustained monomorphic ventricular tachycardia. Similar to the QRS complexes of the tachycardia, the premature ventricular complexes have an initial Q wave in leads II, III, and aVF (↔), indicating that they originate from the inferior wall and are conducted away from this area, hence the Q wave. This is consistent with the fact that they are likely originating from the inferior wall scar due to the old inferior wall myocardial infarction. ■

Core Case 56

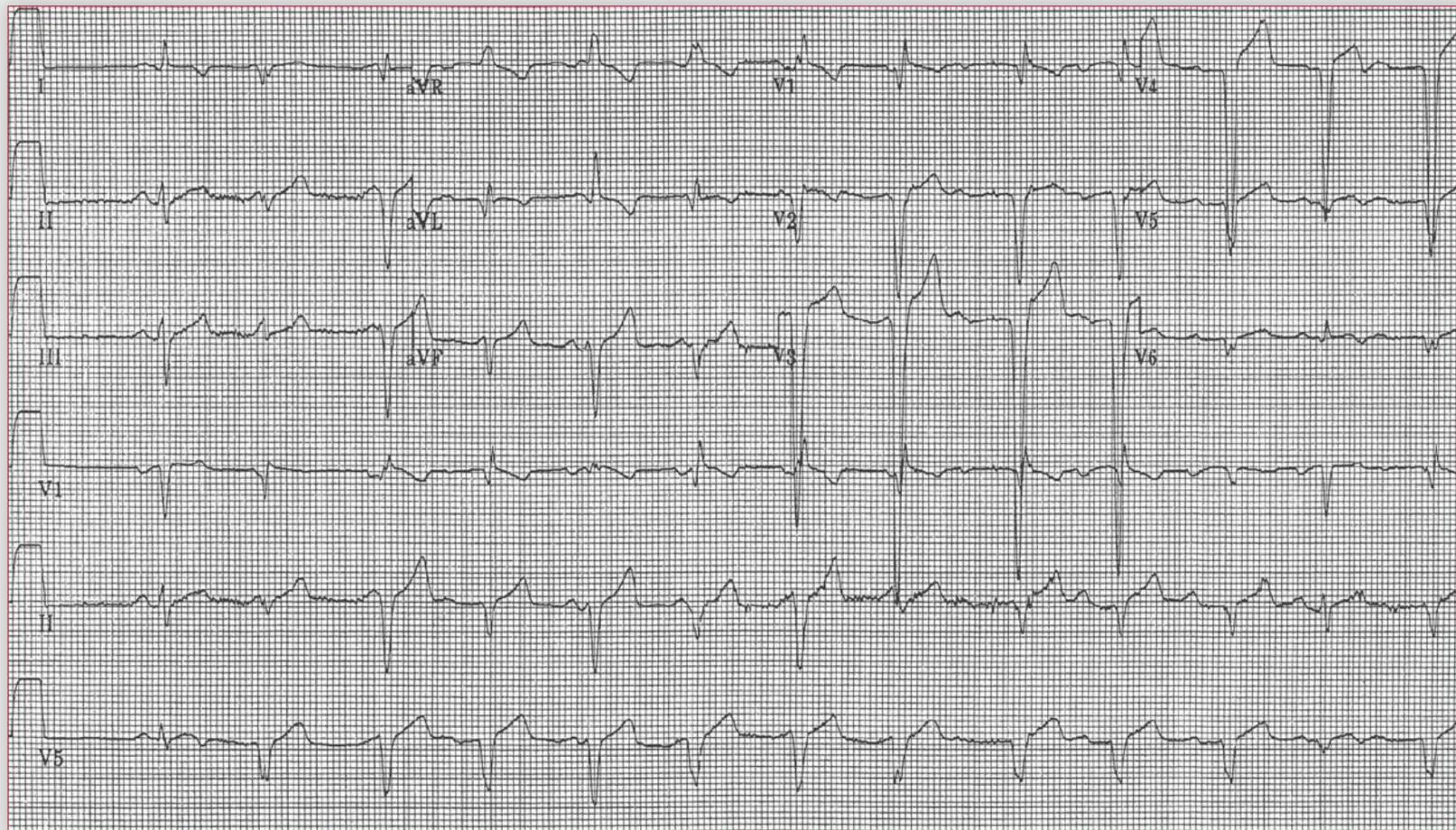
A 60-year-old woman with a history of a nonischemic cardiomyopathy presents to the emergency department because of palpitations that have been present for the past 6 hours. She notes that her pulse rate is more rapid than usual.

ECG 56A



Physical examination reveals a pulse of 112 bpm and irregular heart sounds with a murmur of mitral regurgitation, but is otherwise unremarkable. An ECG is obtained (ECG 56A). The physician notes that the ECG is abnormal and asks for a second tracing (ECG 56B).

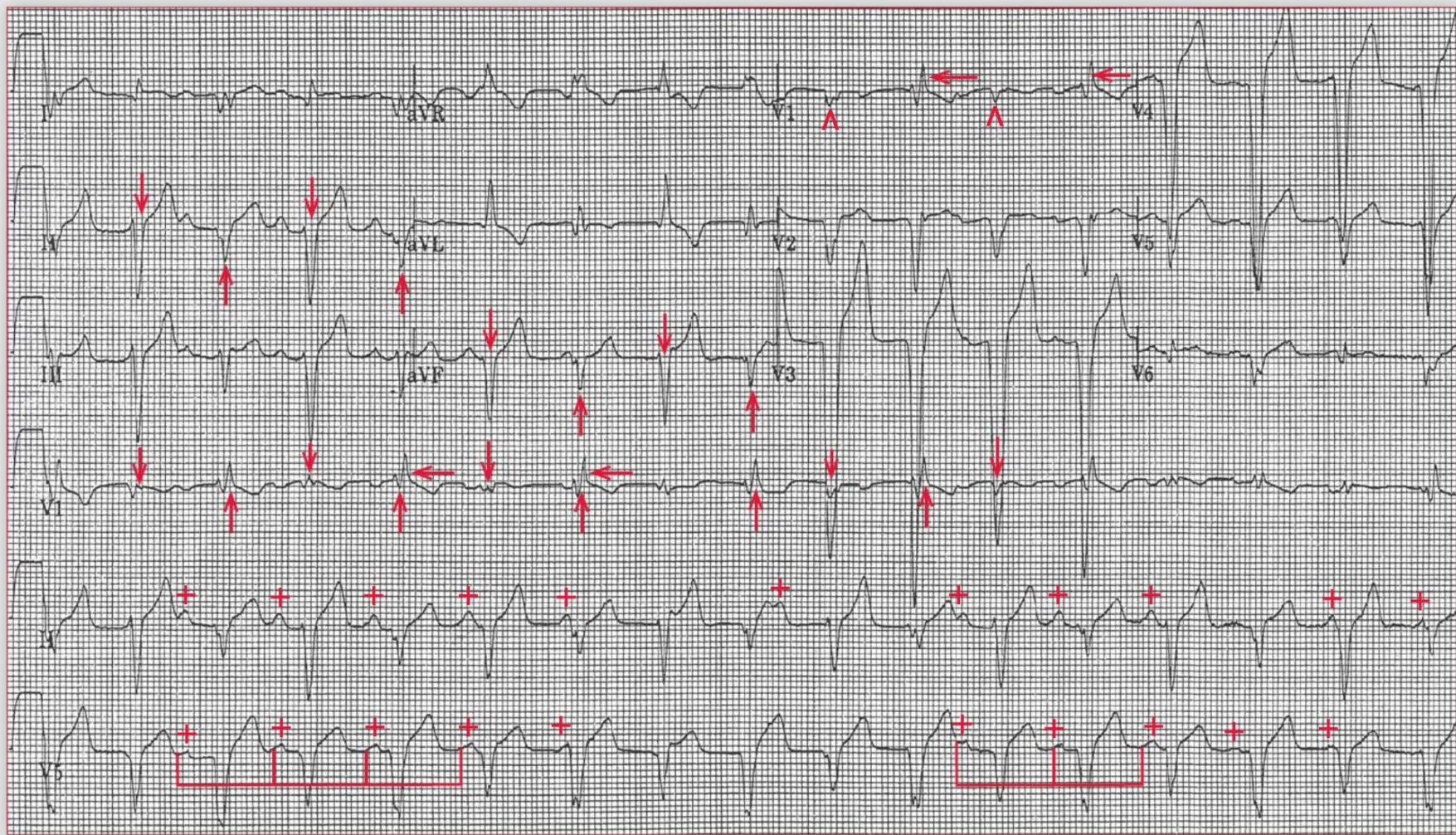
ECG 56B



What abnormality is notable in ECG 56A?

Is the rhythm normal?

Is not, what arrhythmia is present?



ECG 56A Analysis: Sustained bi-directional ventricular tachycardia

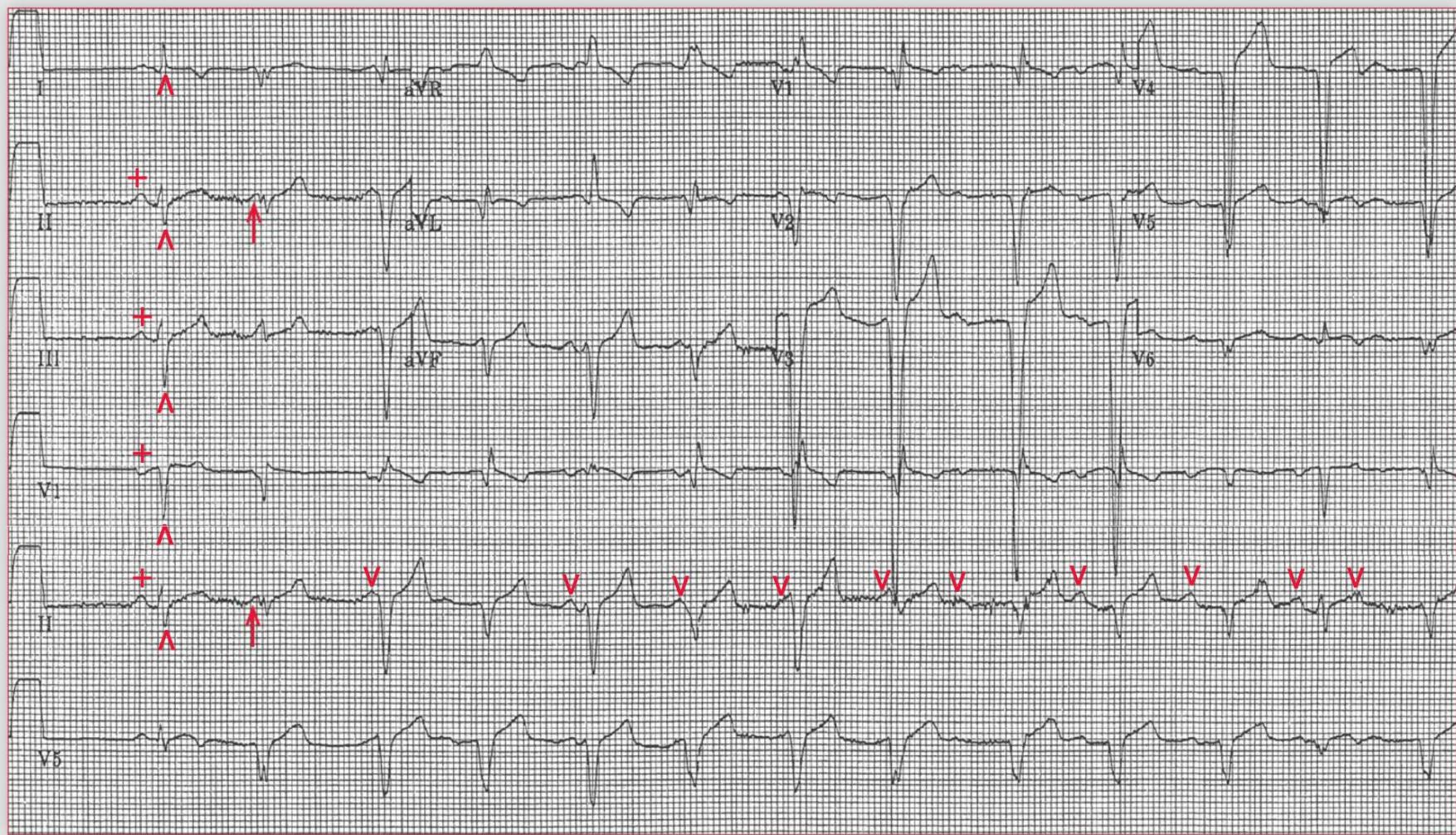
ECG 56A shows a regular rhythm at a rate of 112 bpm. P waves (+) can be seen, and there is a stable PP interval (◻) with an atrial rate of 96 bpm. Although P waves are not always seen, because they are within the QRS complex or T waves, the P waves that are noted occur at a regular interval. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. However, there is no relationship between the P waves and the QRS complexes (*ie*, there are variable PR intervals). Therefore, AV dissociation is present. Since the ventricular rate is faster than the atrial rate, the AV dissociation is the result of an accelerated rhythm.

The QRS complex duration is increased (0.16 sec). However, there are beat-to-beat changes in QRS complex morphology and axis. The odd-numbered complexes (↓) have an extreme left axis, between -30° and -90° (positive QRS complex in lead I and negative QRS complex

in leads II and aVF). These complexes have a left bundle branch block (LBBB)-like morphology, with a QS morphology in lead V1 (↖); however, the morphology is not typical for an LBBB. The even-numbered complexes have an indeterminate axis, between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). In lead V1 these complexes have an RSR' morphology (←), suggestive of a right bundle branch block (RBBB), although the morphology is not typical for an RBBB. The presence of AV dissociation associated with wide QRS complex tachycardia is characteristic of ventricular tachycardia. Further support for a ventricular origin of the tachycardia is that every other QRS complex has an indeterminate axis and the QRS complexes do not have a typical RBBB or LBBB morphology. The beat-to-beat changes in axis and morphology are consistent with a diagnosis of bi-directional ventricular tachycardia.

continues

Podrid's Real-World ECGs



ECG 56B Analysis: Normal sinus rhythm, bi-directional ventricular tachycardia

In ECG 56B the first QRS complex is narrow (\wedge) and is preceded by a P wave (+), with a PR interval of 0.18 second. The QRS complex has a normal duration and morphology. Hence this is a normal sinus complex. There is a P wave (\uparrow) before the second QRS complex, with a PR interval that is shorter than in the first QRS complex. In addition, the second QRS complex has a different morphology. This is probably a fusion complex (*ie*, resulting from fusion between a ventricular complex and ventricular activation via the normal AV node–His–Purkinje system). Thereafter, there are P waves (\wedge) that are dissociated from the QRS complex. As with ECG 56A, there are changes in QRS complex morphology (although not always beat-to-beat) and the two morphologies are the same as those seen in ECG 56A. The presence of AV dissociation with a fusion complex is diagnostic for ventricular tachycardia. The beat-to-beat changes in QRS complex

morphology are characteristic of bi-directional tachycardia. In contrast to bi-directional tachycardia, which occurs in digoxin toxicity and is a junctional tachycardia with alternating conduction through the right and left bundles or alternating left anterior and left posterior fascicular block, bi-directional ventricular tachycardia is due to a reentrant focus within the ventricular myocardium with probably different points of exit from the circuit into the ventricular myocardium. This may be the result of changes in myocardial refractoriness. However, the exact mechanism for this type of ventricular tachycardia is not certain. Bi-directional ventricular tachycardia is most often associated with conditions in which there is a severe abnormality of the myocardium, including acute myocarditis (especially fulminant myocarditis), decompensated heart failure, and acute myocardial infarction. ■

Notes

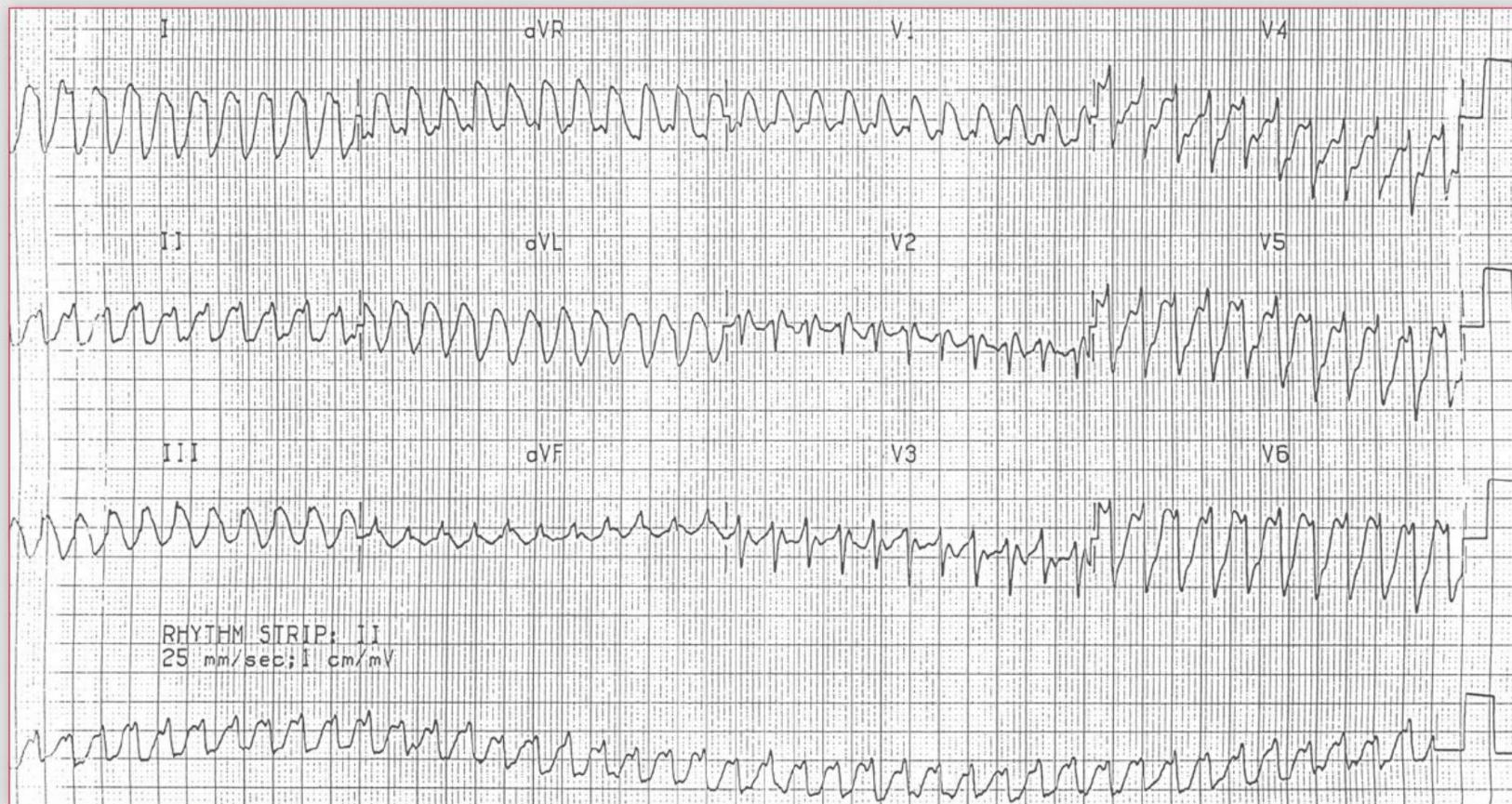
The following ECG is from a 74-year-old patient who was admitted to the intensive care unit with an ST-segment-elevation myocardial infarction. She had received thrombolytic therapy 2 days earlier. The patient, who had a Swan-Ganz catheter in place, was complaining of chest pain and dyspnea in the minutes preceding the ECG

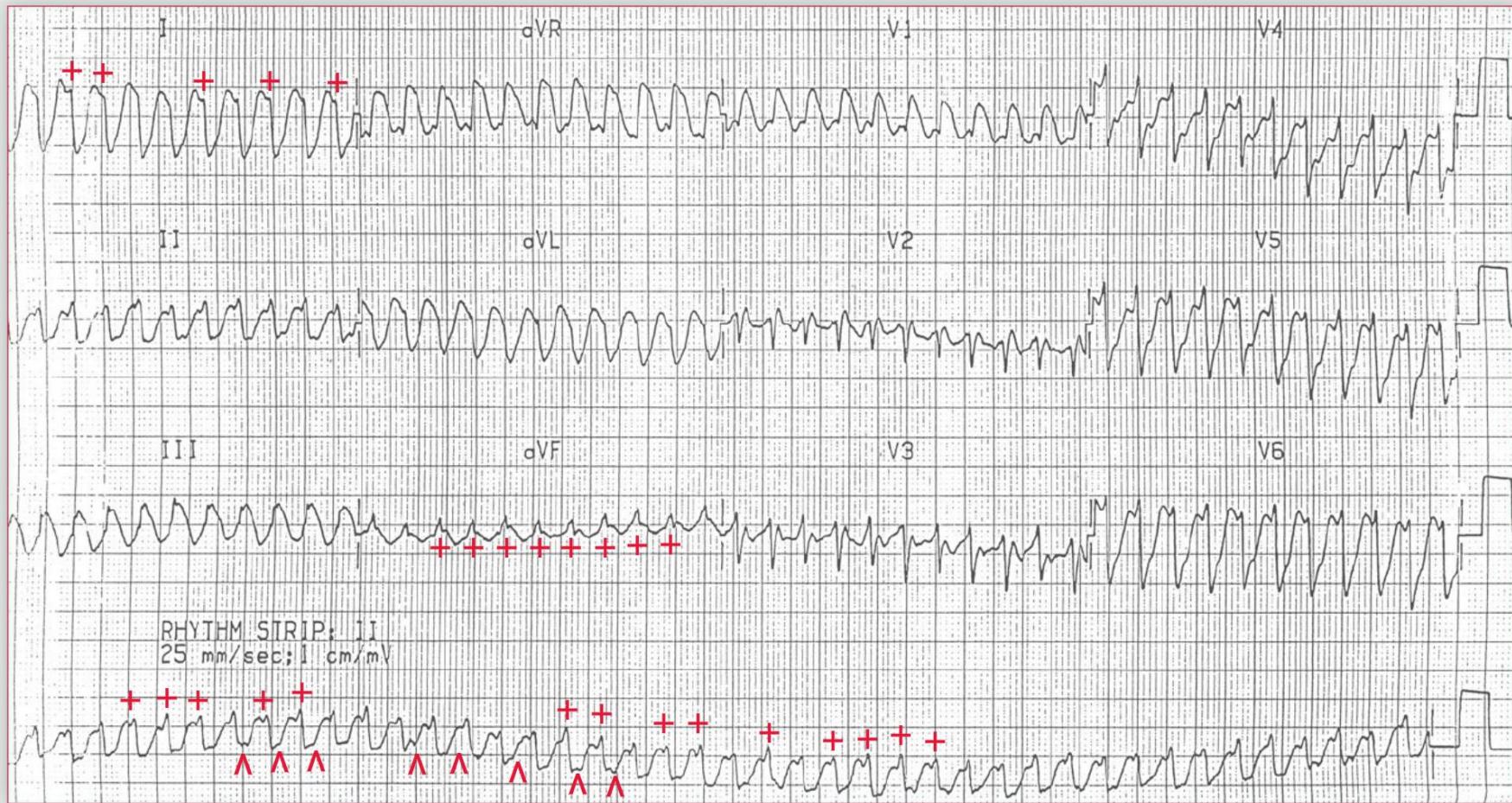
and became acutely unresponsive and pulseless while the ECG was being obtained. Thirty minutes prior to this acute episode the nurse noted a rise in pulmonary capillary wedge pressure from 15 to 25 mm Hg with tall v waves and normal y descents as well as a mixed venous oxygen saturation of 85%, up from 70%.

What is the arrhythmia?

What is the clinical diagnosis?

How would you manage this patient acutely?





ECG 57 Analysis: Sustained monomorphic ventricular tachycardia (ventricular flutter)

There is a regular rhythm at a rate of 270 bpm. The QRS complex duration is 0.16 second; the QRS morphology is abnormal and the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are only two arrhythmias that present with a regular rate over 260 bpm (*ie*, atrial flutter with 1:1 conduction or ventricular tachycardia, which at this rate is often termed ventricular flutter). There is no obvious atrial activity. There is subtle variability in the morphology of the QRS complex (+) and ST-T waves (Δ), most apparent in the lead II rhythm strip as well as leads I and aVF (+). These are features seen with sustained ventricular tachycardia. The morphology of the QRS complexes is similar; hence this is monomorphic ventricular tachycardia. At this rate (> 260 bpm), ventricular tachycardia is termed ventricular flutter.

Immediate defibrillation is indicated when the arrhythmia is associated with hemodynamic compromise, as in this patient who became pulseless. Importantly, cardioversion (which is the delivery of a low-energy shock synchronous with the QRS complex) should not be used as at this rate it is not possible to distinguish between the QRS complex and the T wave, and the shock could be delivered on top of the T wave. Defibrillation is the delivery of a very high-energy shock not synchronized to the QRS complex. In addition, CPR and advanced cardiovascular life support should be performed, including airway maintenance and oxygenation (*ie*, intubation), chest compressions, and administration of epinephrine (+/- vasopressin) and anti-arrhythmic therapy at the appropriate time intervals. If a pulse is restored, then ensuing hypotension can be addressed acutely with vasopressor therapy.

If no other clinical information is available, the history of a recent acute myocardial infarction treated with thrombolytic therapy means that the most likely cause of ventricular tachycardia is scar formation. However, the fact that the ventricular tachycardia rate is this fast suggests the possibility of underlying ischemia, which can shorten the refractoriness of the myocardium and hence cause the ventricular tachycardia rate to be faster. Emergent coronary angiography for further evaluation is warranted. Additionally, an urgent echocardiogram should be performed to assess for a mechanical complication of myocardial infarction, including papillary muscle rupture, ventricular septal rupture, or free wall rupture.

However, in this patient, the diagnosis can be established based on data from the Swan-Ganz catheter. Both acute papillary muscle rupture with severe mitral regurgitation and ventricular septal rupture with left-to-right shunting will give an abrupt rise in pulmonary capillary wedge pressure (PCWP) due to increased venous return to the left atrium with tall v waves. However, only in the setting of a left-to-right shunt will the mixed venous oxygen saturation increase from a normal value of 70% to 85%. Therefore, this patient has sustained a ventricular septal rupture, which requires urgent surgical repair once the arrhythmia has been treated.

Free wall rupture generally results in tamponade physiology with equalization of left- and right-sided diastolic pressures, a blunted y descent on PCWP tracing, and pulsus paradoxus (*ie*, a drop in systolic blood pressure > 10 mm Hg on inspiration). ■

Core Case 58

A 42-year-old man with no prior cardiac disease presents to the emergency department with acute-onset palpitations and lightheadedness. He is tachycardic but has a normal blood pressure of 120/80 mm Hg. His cardiac exam is notable for a

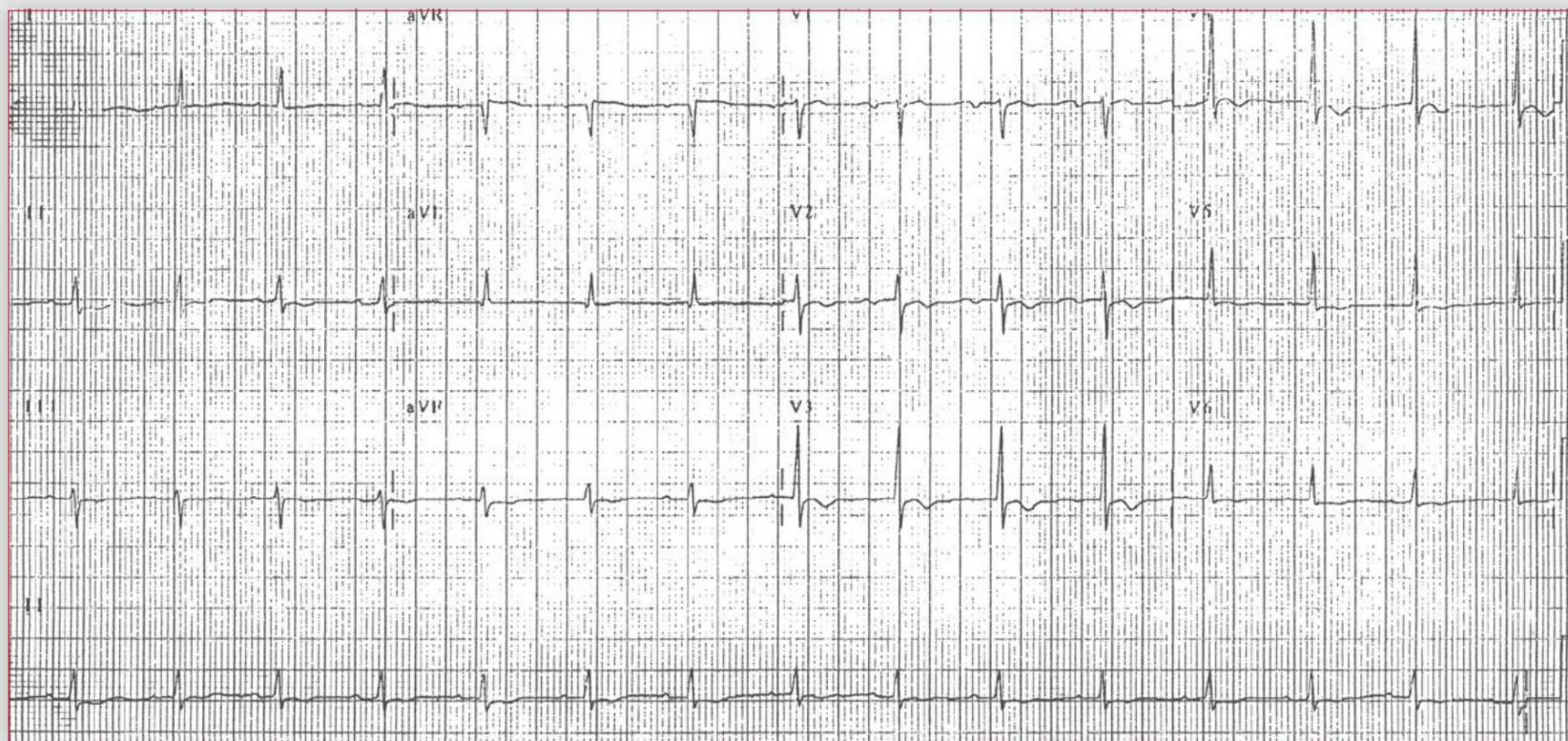
ECG 58A

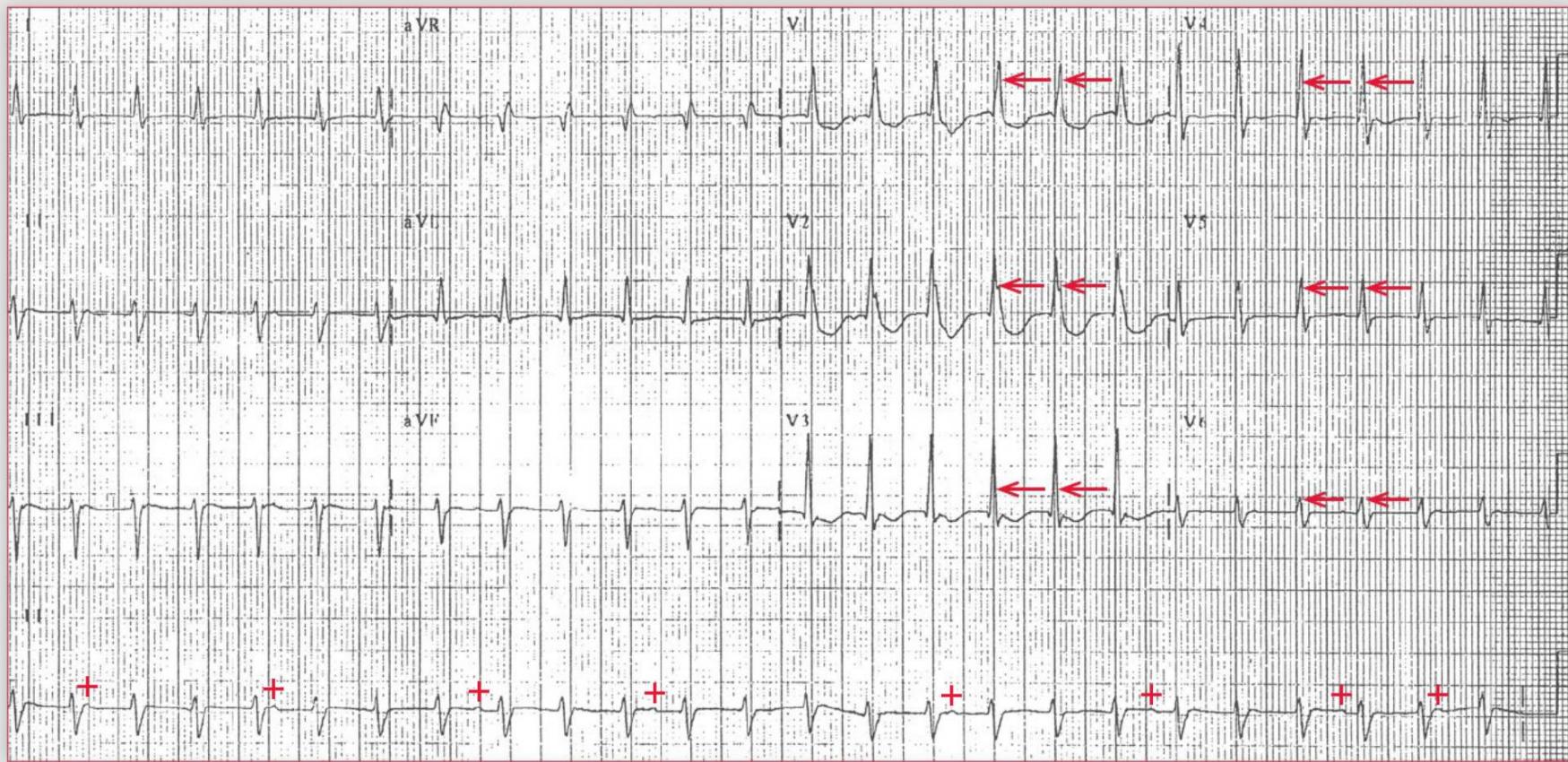


diffuse and laterally displaced point of maximal impulse, but on auscultation there are no murmurs or gallops. An ECG (58A) is obtained because of the tachycardia. This ECG is compared with a previously recorded ECG (58B).

What is the rhythm abnormality seen in ECG 58A?
What is the mechanism of this arrhythmia?

ECG 58B





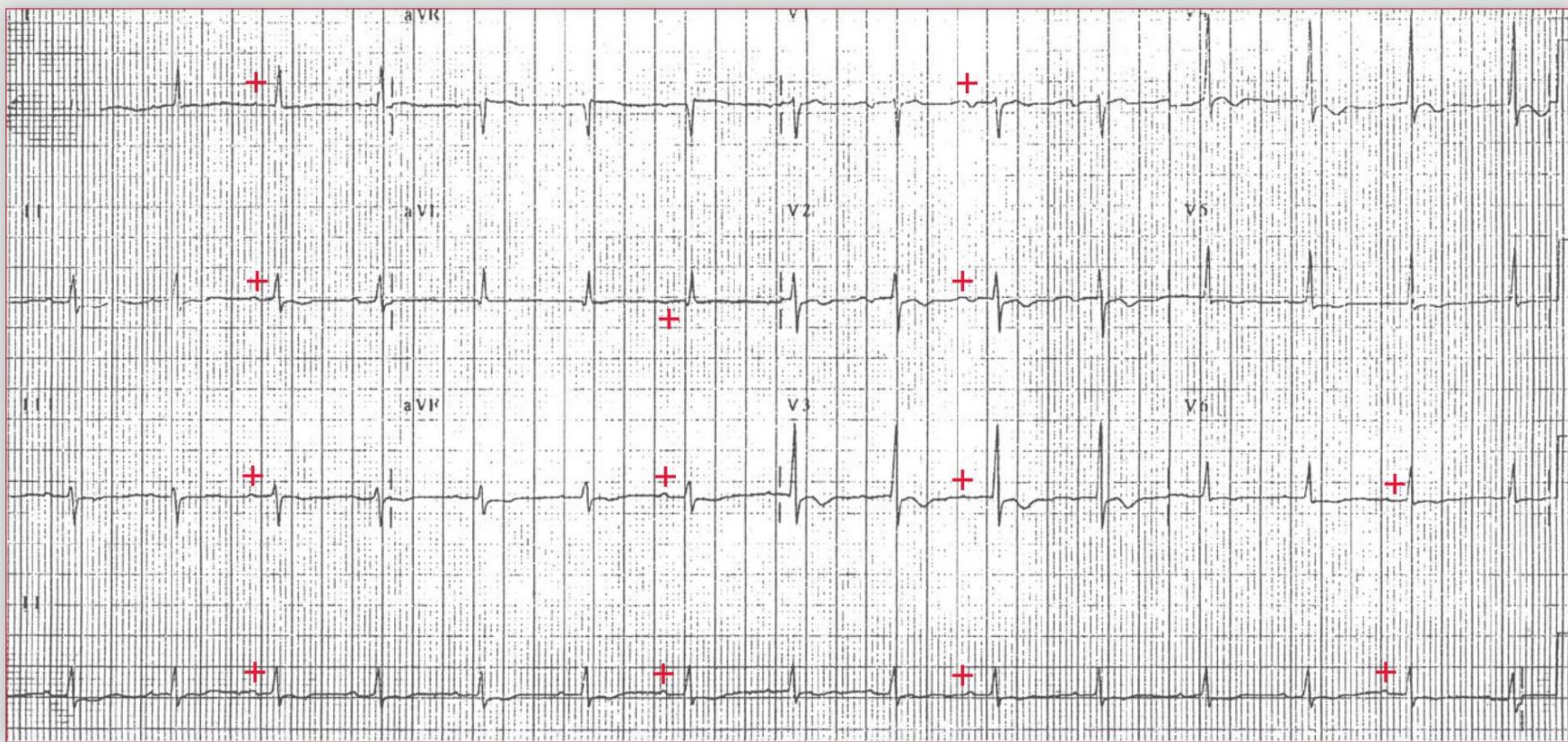
ECG 58A Analysis: Left posterior fascicular ventricular tachycardia (left ventricular tachycardia, verapamil-sensitive tachycardia, Belhassen tachycardia), AV dissociation

The rhythm is regular in ECG 58A at a rate of 152 bpm. The QRS complex duration is 0.11 second. Although the QRS complex has a morphology resembling a right bundle branch block (RBBB), it is not typical for an RBBB morphology as there are tall R waves in leads V1-V5 (\leftarrow) or (almost) positive QRS complex concordance. The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology); this is consistent with a left anterior fascicular block. Noted are P waves (+) that are dissociated from the QRS complexes, particularly obvious in the lead II rhythm strip (*ie*, after the second and fifth QRS complexes, before the ninth QRS complex, after the 11th and

16th QRS complexes, before the 20th and 23rd QRS complexes, and after the 24th QRS complex).

Although the QRS complex duration is only slightly increased, the presence of AV dissociation is generally characteristic of ventricular tachycardia. It is rare for supraventricular tachycardia to have AV dissociation. Situations in which there may be junctional tachycardia with AV dissociation include acute myocardial infarction or a cardiomyopathy. In addition, there is (almost) positive concordance across the precordium, which is another feature associated with ventricular tachycardia.

continues



ECG 58B Analysis: Normal sinus rhythm, counterclockwise rotation (early transition), diffuse nonspecific ST-T wave abnormality

Importantly, the QRS complex morphology should be compared with that present during sinus rhythm, as is seen in ECG 58B. Noted are small P waves (+) before each QRS complex with a stable PR interval (0.20 sec). The P waves are upright in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm at a rate of 74 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is physiologically leftward, between 0° and -30° (positive QRS complex in leads I and aVF and negative QRS complex in lead II). The QT/QTc intervals are normal (360/400 msec).

Hence ECG 58A shows tachycardia with AV dissociation and a QRS complex that is very different than that of sinus rhythm (*ie*, there is a left anterior fascicular block and tall R waves in leads V1-V5, resembling an RBBB pattern). The QRS complex width is only slightly prolonged. These are characteristics associated with fascicular tachycardia, which is ventricular tachycardia that originates in proximity to one of the fascicles within the ventricle. The presence of a left axis

and an RBBB pattern is seen with left posterior fascicular tachycardia or with ablation of the left posterior fascicle. Fascicular tachycardia has also been called left ventricular tachycardia, Belhassen tachycardia or verapamil-responsive tachycardia as this agent often terminates the arrhythmia and prevents recurrence. It usually occurs in patients without structural heart disease and, therefore, tends not to be life-threatening. Therapy may be medical (*ie*, verapamil or a β -blocker) or surgical (*ie*, ablation of the left posterior fascicle).

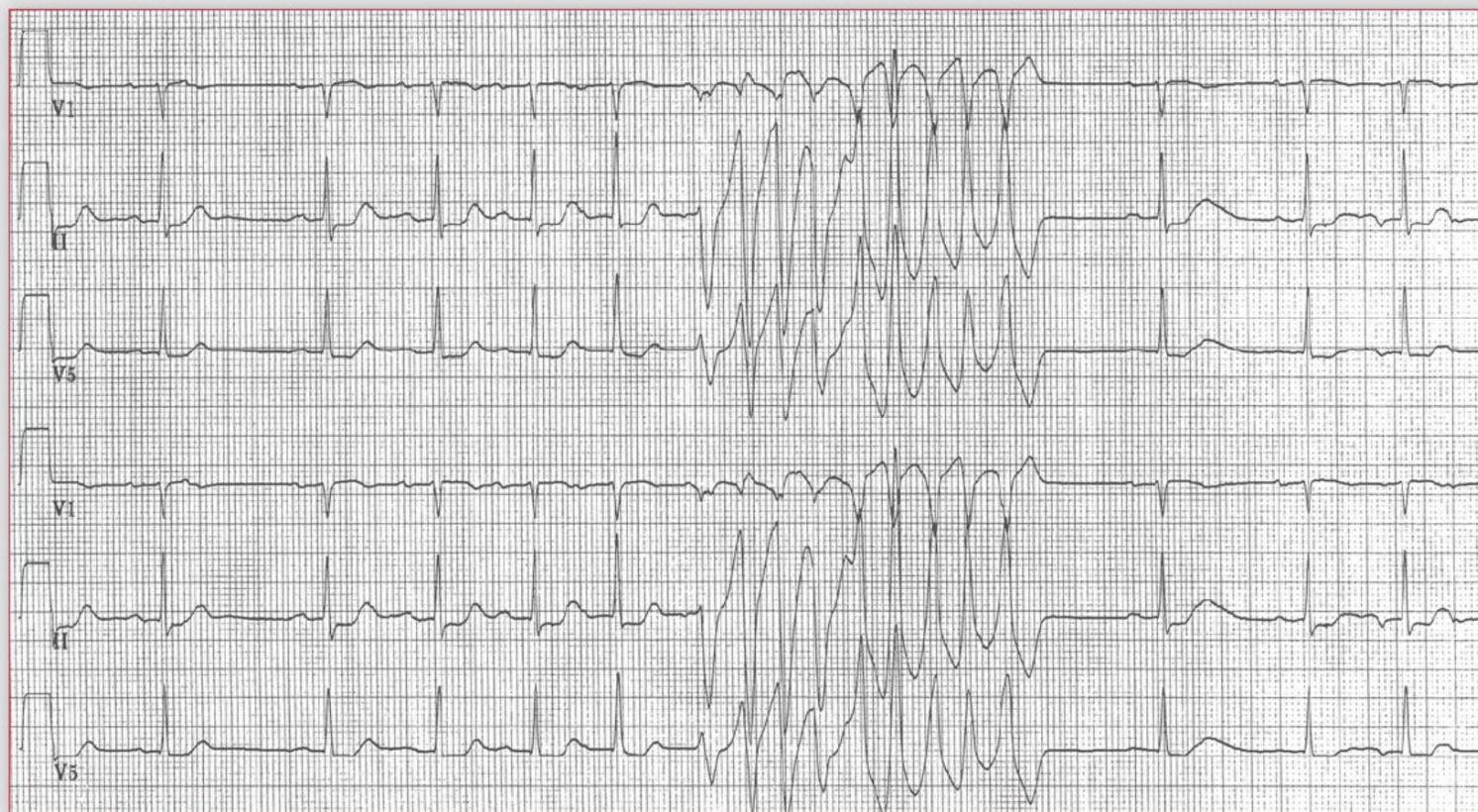
Fascicular tachycardia is a result of a reentrant mechanism involving one of the fascicles (in this case the left posterior fascicle) and the surrounding ventricular myocardium. The QRS complex duration is relatively normal (often > 0.10 but < 0.12 sec), and AV dissociation is present. The most common type is left posterior fascicular tachycardia. The QRS complex has a morphology that resembles an RBBB (with tall R waves across the precordium) and a marked left axis (left anterior fascicular block). ■

Notes

A 56-year-old man with diabetes, hypertension, and an active smoking history presents with acute-onset substernal chest pressure while at rest. He denies any history of angina or exertional dyspnea. Inferoapical ST-segment depressions are noted on the initial ECG. Cardiac biomarkers are initially negative. The patient's symptoms are controlled with nitroglycerin, and the ST-segment

depression resolves. He is started on aspirin, clopidogrel, intravenous unfractionated heparin, and an oral β -blocker and is admitted to the telemetry unit with plans for cardiac catheterization in the morning. Subsequently, the patient has recurrent chest pressure and intermittent episodes of dizziness. The following rhythm strips are obtained from telemetry.

**What is the arrhythmia?
How would you manage
this patient?**





ECG 59 Analysis: Nonsustained polymorphic ventricular tachycardia
with a normal QT interval, normal sinus rhythm with first-degree AV block,
premature atrial complex, ischemic ST-segment depression

The initial portion of the rhythm strips shows a narrow QRS complex (0.08 sec). The rate is 84 bpm. There is a P wave (+) in front of the QRS complex, and the P wave is positive in leads II and V5. Hence this is likely a sinus rhythm. The PR intervals of the first, second, third, and fourth QRS complexes are the same (0.24 sec). The fifth QRS complex (↓) is early and is preceded by a P wave (*) that is also early; hence this is a premature atrial complex. The long interval between the first and second QRS complexes (↔) is the result of a blocked premature atrial complex; a premature P wave (Λ) can be seen on the T wave of the first QRS complex. The QT/QTc intervals are normal (360/430 msec).

In the middle of the rhythm strip there is a wide complex tachycardia (Π) with an irregular rate and with QRS complexes that are variable in morphology, including a change in axis. This is termed polymorphic ventricular tachycardia, and it is nonsustained. Following the run of polymorphic tachycardia are four narrow QRS complexes that are preceded by a P wave; the PR interval is stable. The first two QRS complexes have a P wave (●) with the same morphology as seen in the initial QRS complexes before the ventricular tachycardia; these are sinus complexes. The next-to-last QRS complex is preceded by a negative P wave (▲); this is a premature atrial complex. It should also be noted that there is ST-segment depression (↑) in leads II and V5, diagnostic for ischemia.

There are two subtypes of polymorphic ventricular tachycardia, which depend on the QT/QTc intervals of a sinus complex:

- If the baseline QT interval of a sinus complex is normal, as in this case, it is termed polymorphic ventricular tachycardia and the most common etiology is ischemia. Another uncommon etiology is familial catecholaminergic polymorphic ventricular tachycardia, which is due to a genetic abnormality of either the ryanodine receptor or the calsequestrin 2 gene.
- Polymorphic ventricular tachycardia that occurs in association with prolongation of the baseline QT interval of a sinus complex is called torsade de pointes (twisting of points). The prolongation of the QT interval may be either acquired (*ie*, the result of a drug) or congenital (*ie*, the result of a genetic abnormality that produces a channelopathy).

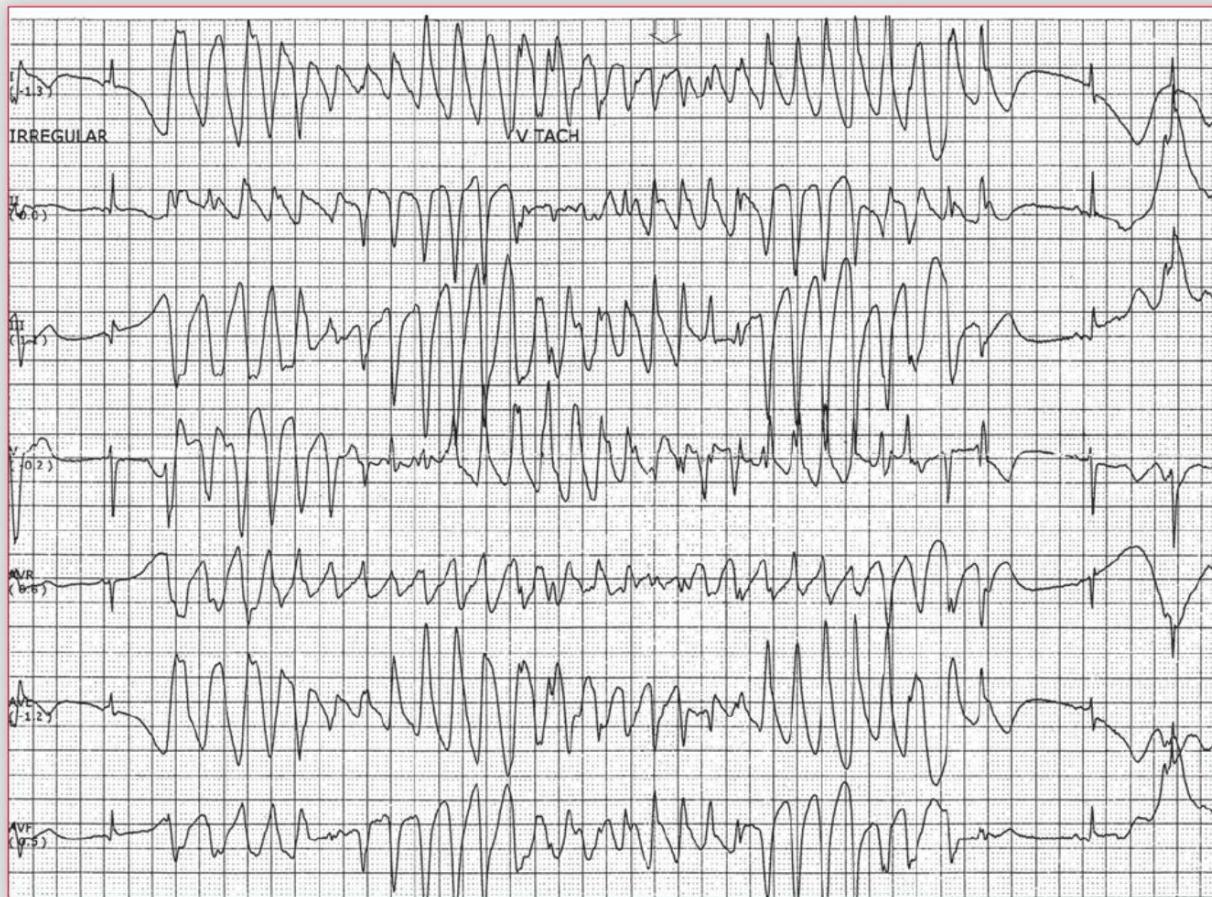
In this case, the patient is having recurrent ischemia, as is evident by the clinical story, the presence of ST-segment depression, and intermittent nonsustained polymorphic ventricular tachycardia. An intravenous β -blocker should be administered to reduce myocardial demand and ischemia, thereby reducing the risk for recurrent arrhythmia. Intravenous nitroglycerin is also indicated to relieve ischemia as a result of a decrease in venous return and a reduction in left ventricular wall stress. Given the unstable nature of polymorphic ventricular tachycardia, the patient should undergo urgent coronary angiography as well as revascularization if appropriate. ■

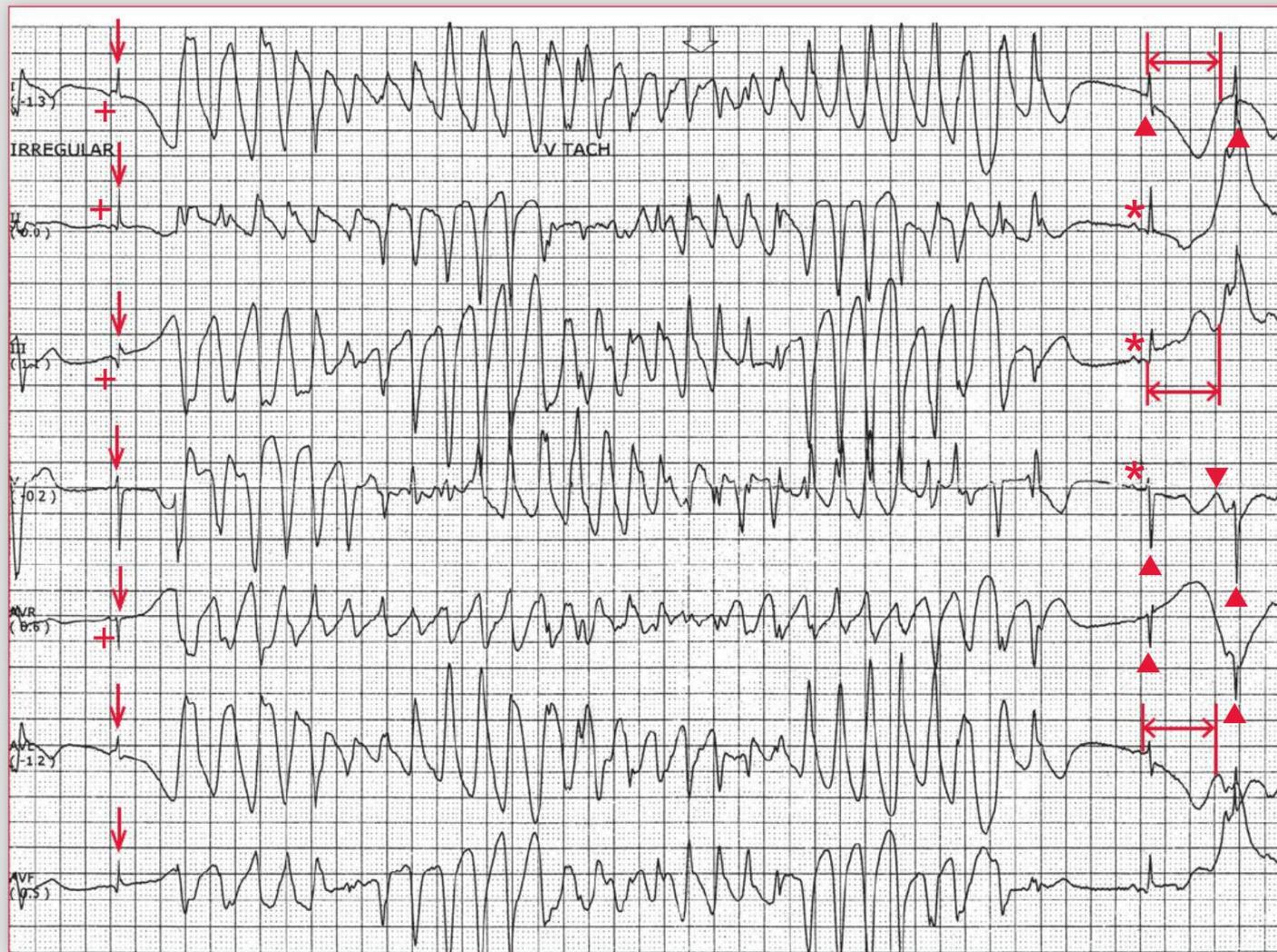
Notes

A 22-year-old man with no previous cardiac history, but with a history of a seizure disorder for the past 4 years, presents to the emergency department after an unwitnessed syncopal episode. He is admitted to the hospital and placed on telemetry. A few hours into his hospitalization, he experiences a recurrent syncopal episode, during which the following telemetry strip is recorded.

What is the arrhythmia?

What is the clinical cause of his arrhythmia?





ECG 60 Analysis: Torsade de pointes (nonsustained polymorphic ventricular tachycardia associated with a long QT interval)

The first QRS complex (↓) has a normal duration and is preceded by a P wave (+). This is followed by an episode of a wide QRS complex rhythm with marked variability of QRS morphology and a change in axis. This is polymorphic ventricular tachycardia. The last two QRS complexes on the ECG (▲), which are identical to the first QRS complex (↓), have a normal duration and are preceded by a P wave (*); therefore, these are supraventricular complexes, likely sinus in origin. Although the QT interval of the first QRS complex (↓) is difficult to establish as the T wave is interrupted by the onset of the polymorphic ventricular tachycardia, the QT interval (↔) of the first narrow complex after the arrhythmia can be measured and it is prolonged (600 msec). Therefore, the polymorphic ventricular tachycardia, which is associated with QT prolongation (of the sinus complex), is termed torsade de pointes. Of note is that there appears to be a U wave superimposed on the T wave (▼), best seen in lead V1 (fourth line). This is referred to as a QT-U wave and is most commonly seen with congenital QT prolongation. Along with the history of several years of seizures, which are often caused by undiagnosed torsade de pointes, this ECG pattern is typical of a congenital long QT syndrome.

A congenital long QT syndrome is due to a channelopathy involving a membrane potassium, sodium, or calcium channel. Although more than 10 genetic variations have been identified, the most common abnormality is a gene involving the potassium channels, resulting in the long QT (*LQT1* and *LQT2*). In congenital QT prolongation (*ie*, *LQT1* or *LQT2*), torsade de pointes is often precipitated by exercise or an increase in sympathetic activity. With the increase in heart rate the QT interval fails to shorten appropriately and may even lengthen, increasing the risk for torsade. In addition, sympathetic stimulation can increase the frequency and amplitude of early after-depolarizations (low amplitude membrane oscillations that occur during the prolonged phase 2 of the action potential that causes the QT prolongation), resulting in triggered activity and the occurrence of torsade.

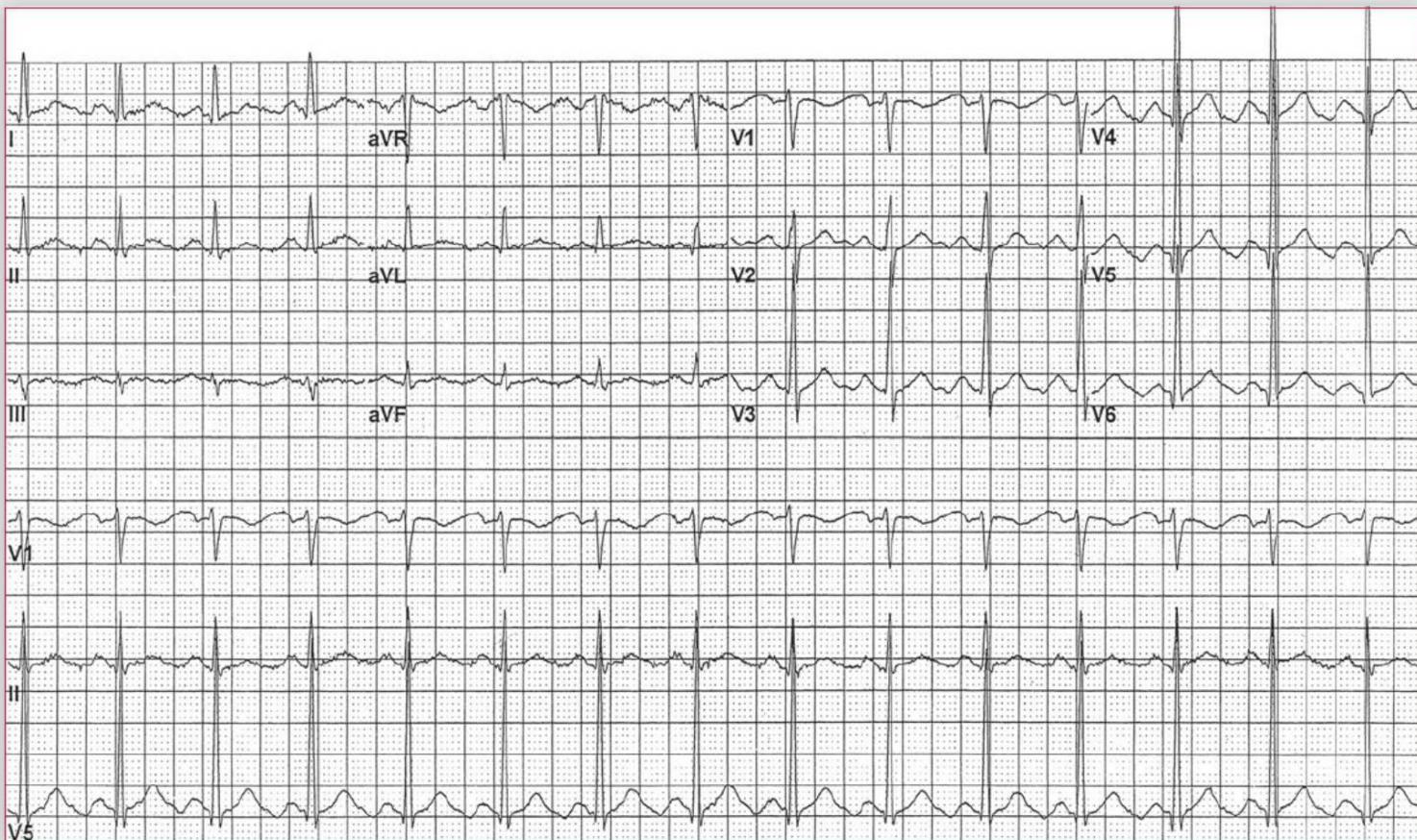
Since sympathetic stimulation and an increase in heart rate are associated with torsade de pointes in congenital long QT syndrome, therapy with a β -blocker is often acutely effective for preventing recurrent torsade. ■

Core Case 61

A 35-year-old man on chronic methadone therapy for treatment of a traumatic back and hip injury is brought to the emergency department by his brother because of a change in mental status. Although the patient is oriented, he does state that he has been taking two to three times his normal

dose of methadone because of more severe pain. He is admitted to the hospital for observation and is placed on telemetry. A routine admission ECG is obtained (ECG 61A). Several hours after admission premature ventricular complexes are noted on his telemetry and house staff are paged urgently.

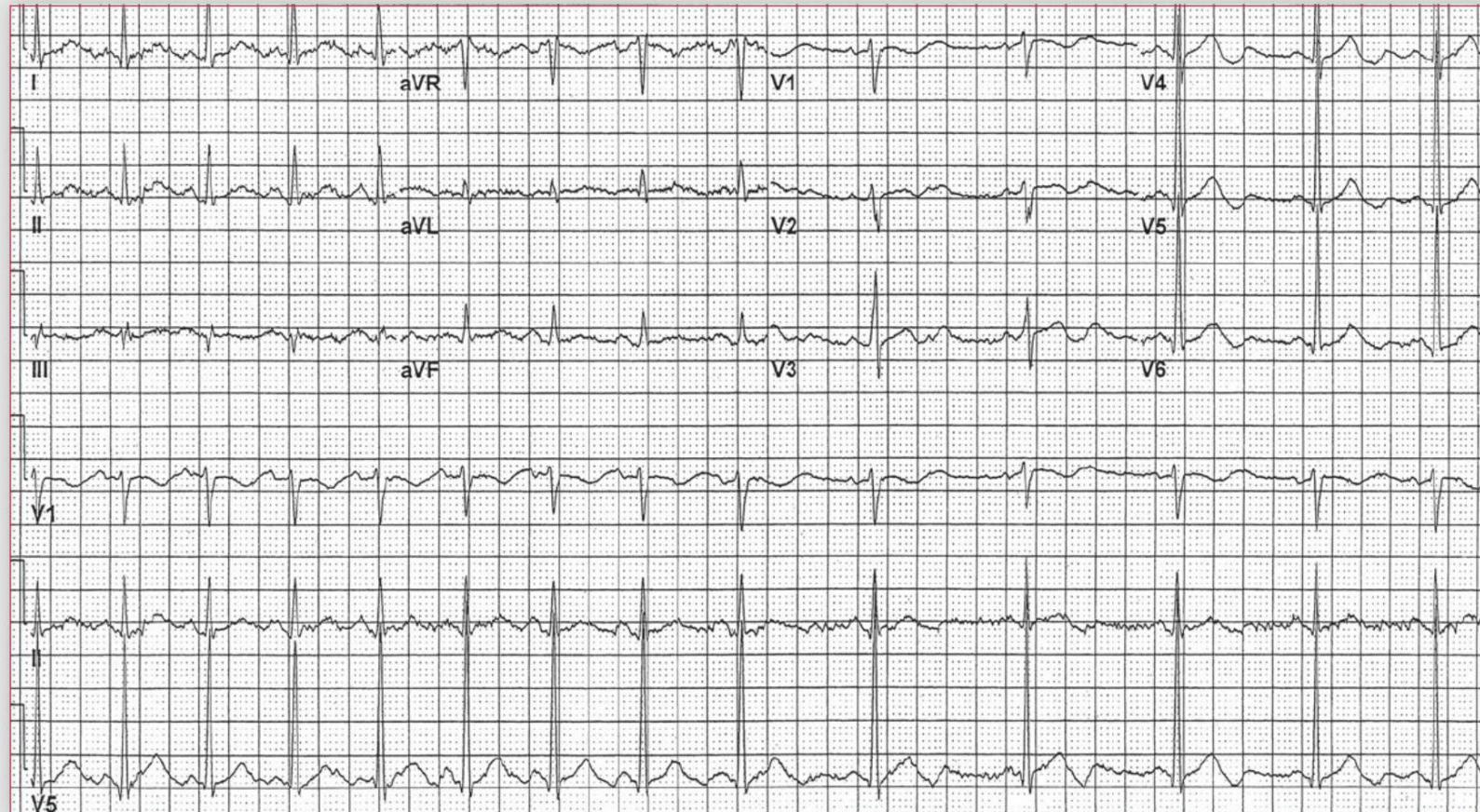
ECG 61A



A second ECG is obtained (ECG 61B). Shortly thereafter, telemetry shows more worrisome abnormalities and rhythm problems (ECG 61C). Methadone was discontinued and 3 days later the man's mental status was normal. An ECG was obtained (ECG 61D).

Is the baseline ECG (61A) normal?
What can be seen on telemetry (ECG 61C)?
What is the cause of the abnormality?
What therapy is indicated?
What does ECG 61D show?

ECG 61B

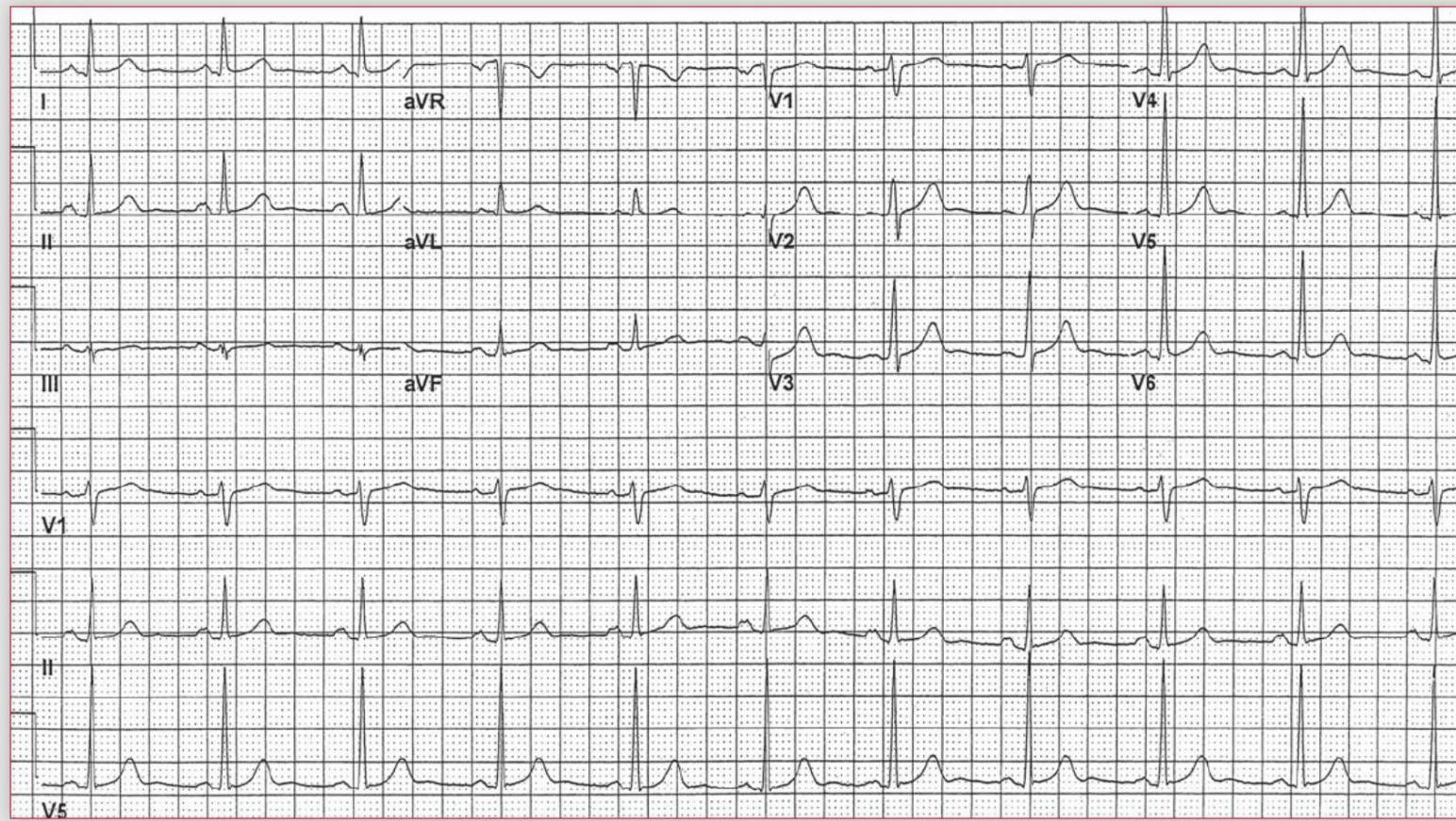


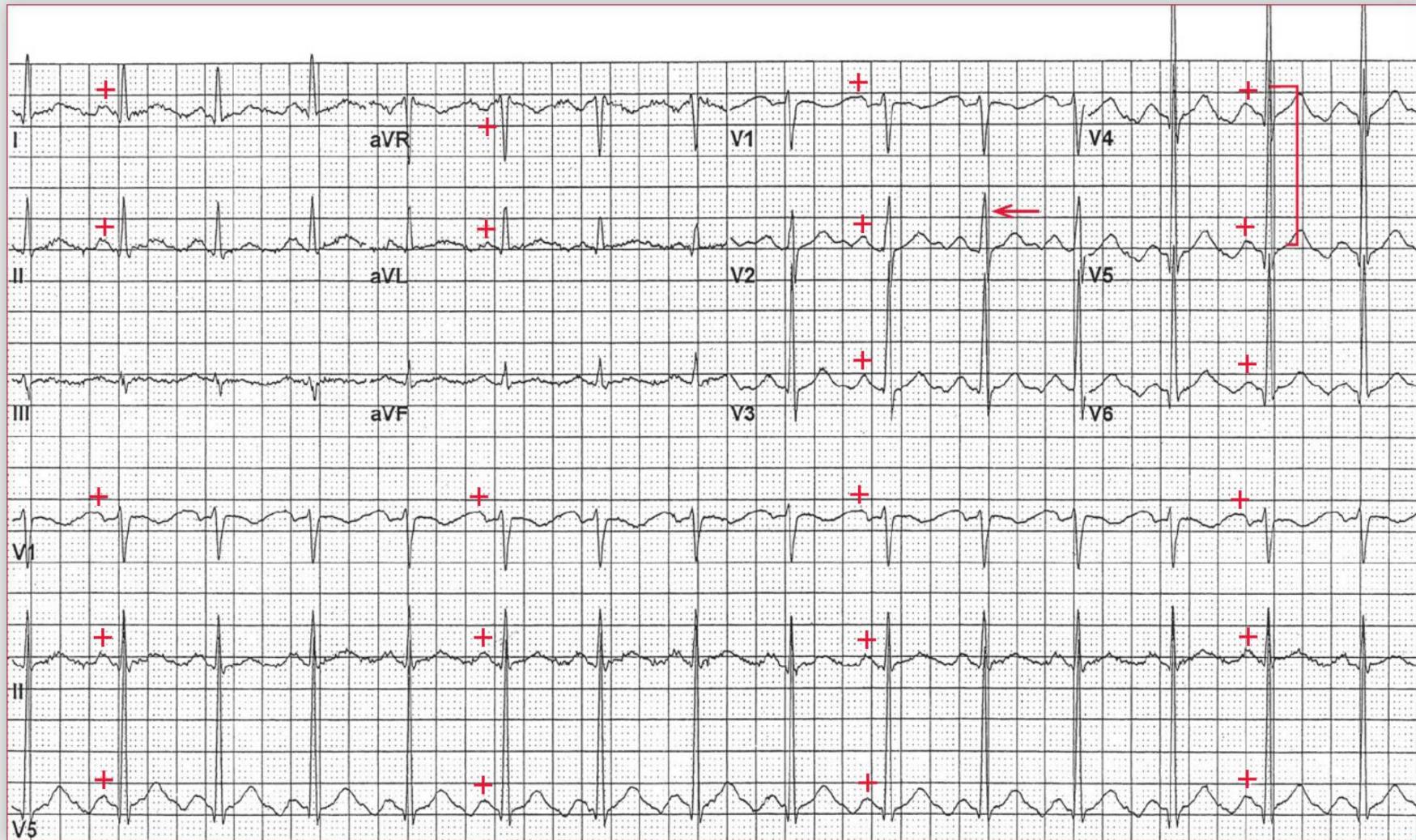
Core Case 61

ECG 61C



ECG 61D





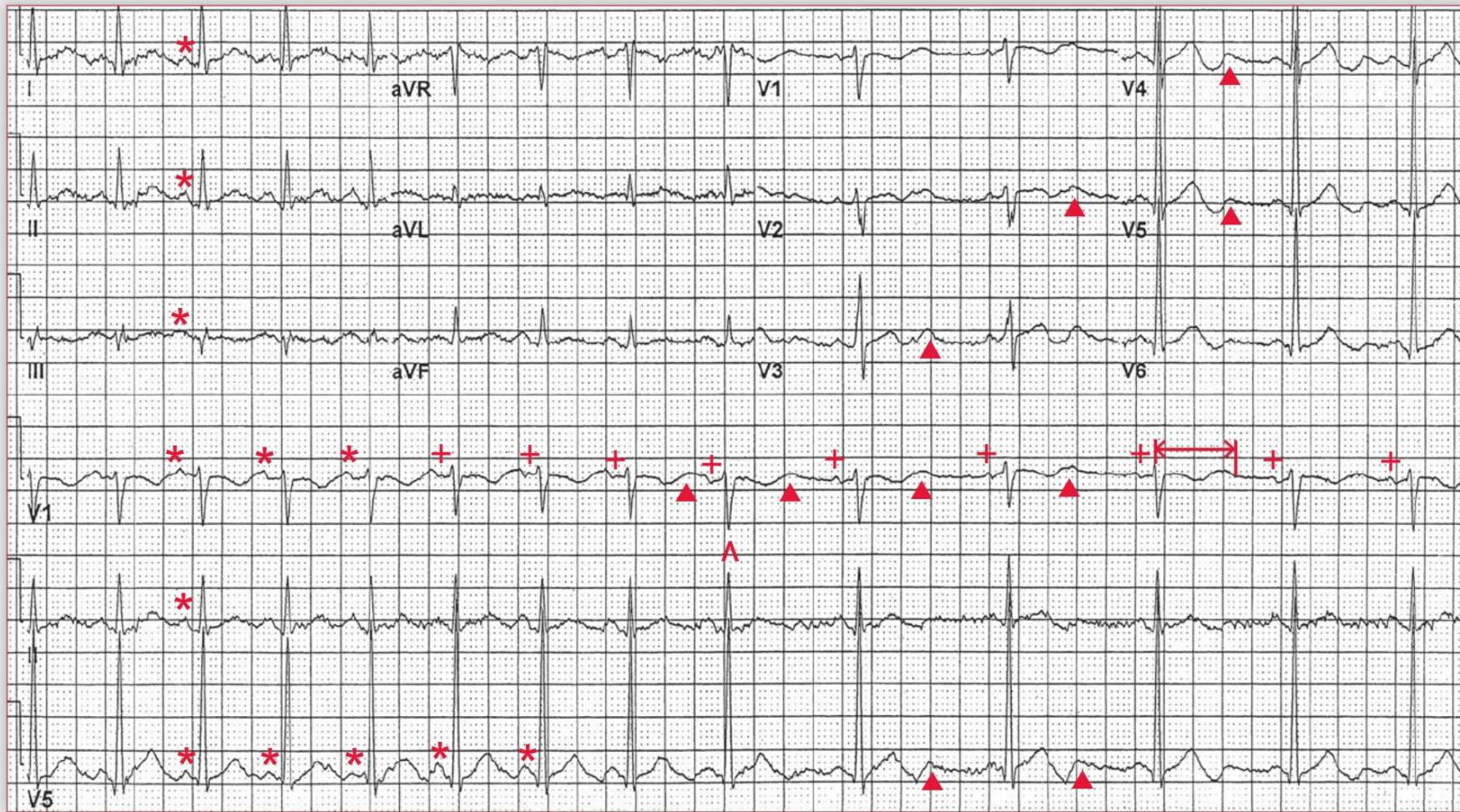
ECG 61A Analysis: Normal sinus rhythm, suggestion of a left atrial abnormality (hypertrophy), possible left ventricular hypertrophy

In ECG 61A the rhythm is regular at a rate of 96 bpm. There is what appears to be a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Therefore, this appears to be a normal sinus rhythm. The P waves are very unusual, however, as they are very broad and tall, especially in leads V1-V6. This suggests left and possibly right atrial hypertrophy (or abnormality).

The QRS complex duration is normal (0.08 sec) and there is a normal axis, between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (400/500 msec). The QRS

complex morphology is normal, although there is very tall voltage in lead V5 (30 mm) (]), consistent with left ventricular hypertrophy. However, the tall QRS voltage may be consistent with a patient of young age, who is thin and has no lung disease. In addition, there is early transition or counterclockwise rotation, with a tall R wave in lead V2 (←). This is due to an axis shift in the horizontal plane and is determined by imagining the heart as viewed from under the diaphragm. When the heart's electrical axis is rotated in a counterclockwise direction, the left ventricular forces develop earlier and are prominent in the right precordial leads.

continues



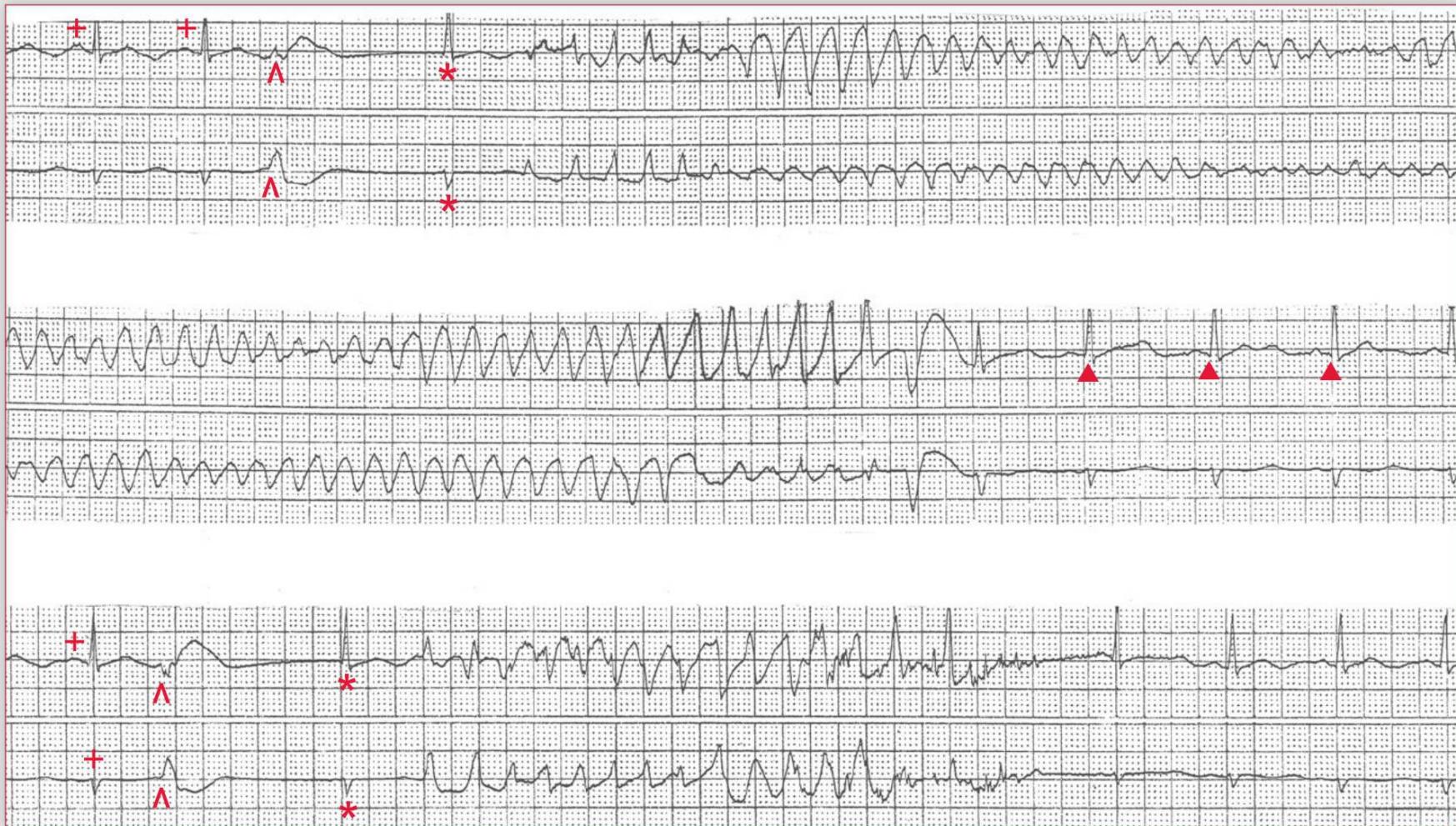
ECG 61B Analysis: Normal sinus rhythm, prolonged QT interval, abnormal T waves

ECG 61B shows an irregularly irregular rhythm as a result of gradual slowing and then acceleration of the rate. The average rate is 78 bpm. The QRS complex duration, axis, and morphology are the same as seen in ECG 61A. The first eight QRS complexes are at a rate of about 100 bpm. Similar to what was seen in ECG 61A, it appears that there is a P wave (*) before each QRS complex with a stable PR interval (0.20 sec). The P wave is very broad and is positive in leads I, II, and aVF. Hence it appears that there is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF).

Similar to ECG 61A, there is voltage criteria for left ventricular hypertrophy, with an R-wave amplitude in lead V5 of 27 mm (]).

However, after the eighth QRS complex (Λ) there is a marked slowing of the heart rate. With the slowing of the heart rate the actual P wave becomes apparent (+); it has a normal duration and is associated with a PR interval of 0.16 second. It is now obvious that what was initially believed to be the P wave is actually the end of an abnormal T wave (▲). It can, therefore, be seen that the QT/QTc intervals are markedly prolonged (↔) (600/680 msec).

continues



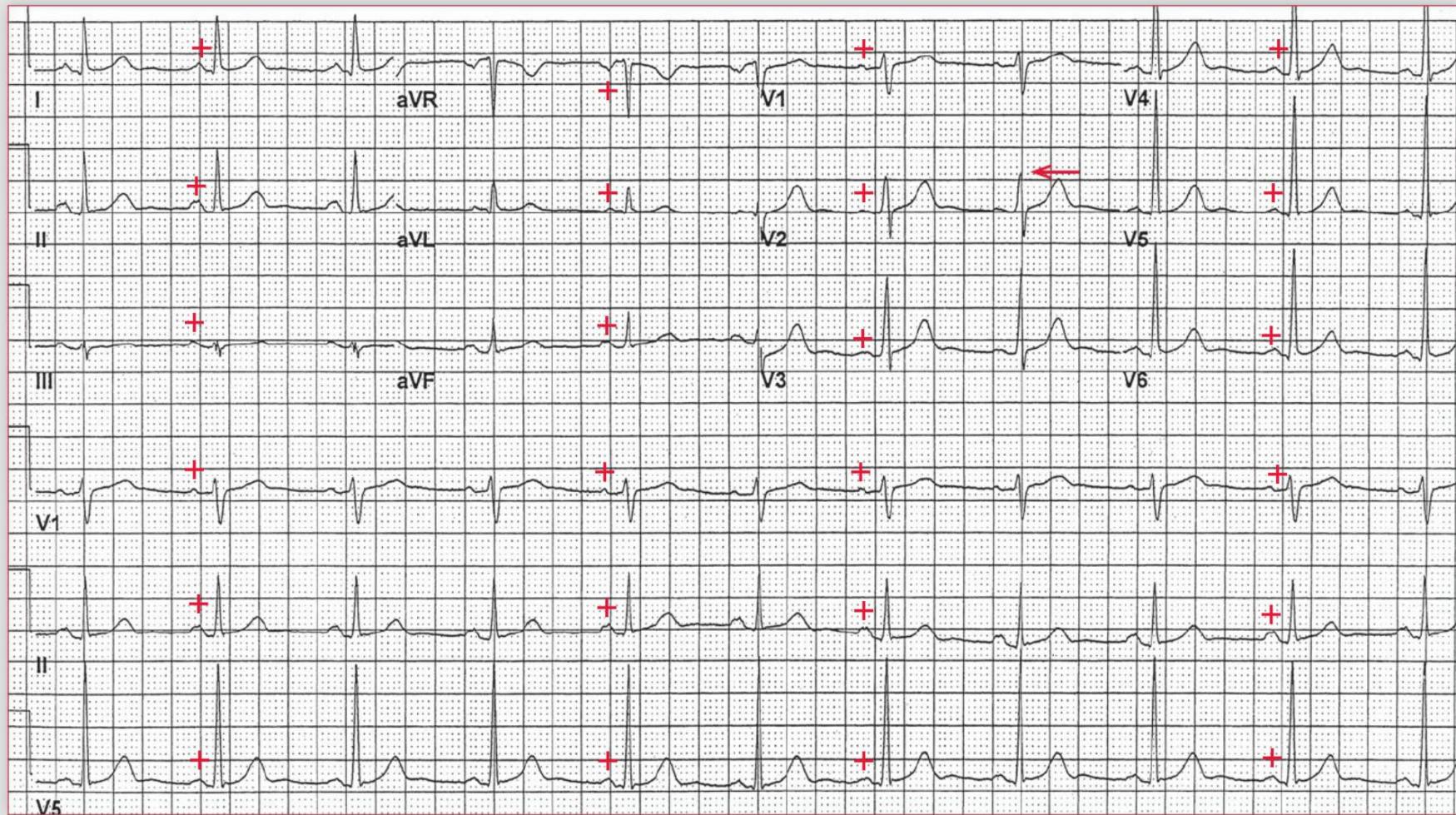
ECG 61C Analysis: Normal sinus rhythm, premature ventricular complexes, torsade de pointes (pause dependent)

ECG 61C shows three rhythm strips. The first two are continuous. On the first rhythm strip there are two narrow QRS complexes that are preceded by P waves (+). Hence these are sinus complexes. The third QRS complex (Λ) is premature, without a P wave, and wide with an abnormal morphology that is different than that of the sinus complexes. This is a premature ventricular complex, after which there is a pause and then another sinus complex (*) followed by rapid tachycardia with a changing QRS complex morphology and axis. This is polymorphic ventricular tachycardia, which terminates spontaneously,

as seen on the second rhythm strip. After termination there are sinus complexes (▲). Polymorphic ventricular tachycardia in association with QT prolongation (of the sinus complex) is called torsade de pointes.

The third strip in ECG 61C shows the same sequence of a sinus complex (+) followed by a premature ventricular complex (Λ) and after the pause a second sinus complex (*). Thereafter, there is a brief episode of torsade de pointes.

continues



ECG 61D Analysis: Normal sinus rhythm, normal ECG

Three days after methadone was discontinued, the patient's ECG returned to baseline (ECG 61D). There is a regular rhythm at a rate of 68 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The PR interval is the same as the PR interval noted in the second half of ECG 61B. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. The QT/QTc intervals are normal (400/425 msec). There is early transition, with a tall R wave in lead V2 (←), as was previously seen.

This patient had an acquired long QT syndrome as a result of an excessive dose of methadone, one of many drugs known to prolong the QT interval. Torsade de pointes associated with the acquired form of long QT syndrome is most commonly associated with bradycardia. Drug-induced or acquired torsade de pointes may also be "pause-dependent" (*ie*, associated with a preceding long-short RR interval). This pattern typically is caused by a premature ventricular complex that is followed by a compensatory pause. The association between pause dependency or bradycardia and drug-induced torsade is thought

to be related to the inverse correlation between the heart rate and QT interval (or the refractory period). (The QT interval decreases as the heart rate increases [shorter RR interval] and lengthens as the heart rate slows [longer RR interval]). This rate-related change in QT interval or membrane refractoriness can further augment QT interval prolongation and membrane refractoriness that result from drugs that prolong the QT interval. This explains why drug-induced torsade de pointes is more commonly seen with bradycardia or is pause dependent.

Treatment for drug-induced or acquired torsade de pointes includes discontinuation of the drug responsible for prolonging the QT interval, correction of any electrolyte abnormalities (especially potassium or magnesium), administration of magnesium (even with a normal serum magnesium), and increasing the heart rate with either overdrive pacing or an intravenous infusion of isoproterenol. The increase in heart rate produces a shortening of the QT/QTc intervals, which can reduce the occurrence of torsade de pointes. Lidocaine therapy may also be effective, particularly by suppressing premature ventricular complexes, eliminating the post-extrasystolic pause seen with premature ventricular complexes. ■

Notes

A 68-year-old man with known coronary artery disease is brought to the hospital by ambulance with complaints of severe crushing substernal chest pain. During ECG testing in the emergency department the man suddenly becomes unresponsive and pulseless.

What is wrong with this ECG?





ECG 62 Analysis: Ventricular fibrillation

The ECG shows a chaotic rhythm without any organized QRS complexes. Instead the waves are irregular in morphology, interval, and amplitude. These are fibrillatory waves and the rhythm is ventricular fibrillation, which is the most common cause of sudden death. The ECG was obtained in an unresponsive patient without a pulse. As there is no cardiac output or blood flow to the brain or other organs, the arrhythmia needs to be reverted as quickly as possible using defibrillation, which is the asynchronous delivery of energy. Usually the maximum output of the defibrillator is used. The only effective therapy for

ventricular fibrillation is prompt defibrillation as this arrhythmia never reverts spontaneously. Defibrillation delivers a high-energy impulse and produces depolarization of the entire ventricular myocardium, making it entirely refractory to electrical stimulation. This eliminates the chaotic electrical activity and allows for a sinus impulse to be reestablished. In general, brain death and other end-organ damage begins about 4 minutes after the onset of ventricular fibrillation, so survival is related primarily to the promptness of defibrillation. ■

Notes